BRAIN.

PART IV., 1904.

Original Articles and Clinical Cases.

ON THE SPINAL CORD DEGENERATIONS IN ANÆMIA.

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THE exact relationship of anæmia to the degenerations in the spinal cord which are comprised under the various terms, "diffuse degeneration," "combined sclerosis," and "subacute combined sclerosis" of the cord, is still a matter about which there is considerable difference of opinion. From a clinical standpoint, anæmia may be associated with degeneration of the spinal cord in at least three ways: (1) it may distinctly precede the spinal changes for a longer or shorter time; (2) it may accompany them from the first, and proceed *pari passu* with them; or (3) it may appear late in the course of the cord affection, just as it often occurs in the later stages of any chronic illness. In the last instance, the anæmia is not as a rule so profound as in the other two. It should be added that in any case the anæmia may from time to time undergo considerable remissions, and occasionally be cured.

Although the subject is not a new one, many cases having been published, as it is still to a certain extent

sub judice, the following cases, which belong entirely to the first group, where anæmia predominates over and precedes the spinal symptoms, and which illustrate both the early stages of the degeneration in the posterior columns of the cord and also their later development, have seemed to me not unworthy of record. I wish to emphasise that this paper deals only with cases in which the cord changes were, so far as one could judge, secondary to antecedent severe anæmia. In two of them disease of the cord was not suspected during life, and in all the anæmia was of considerable duration. It is not, therefore, a question in these cases of anæmia appearing in the course of primary chronic spinal cord disease. The second case is of especial interest, as I was enabled to watch it throughout the whole course of the illness; it presents the cord affection in an early stage, and is further interesting in that the anæmia was cured, and the cord changes had apparently ceased to advance.

With regard to the form of anæmia present in these and similar instances it is now established that it is by no means only, nor even most often, pernicious anæmia. In the published cases there is a lack of exact observations on the blood, and I regret that this fault unavoidably attaches to one of two of the following reports.

The first case was the first example in which the slighter changes in the cord came under my observation, and appeared to represent the earliest stage in the cord affection of anæmia, but as the patient was only under observation during a short and fatal illness this was not certain. The correspondence of the lesion, though in a less advanced form, with that in Case 2, in which, as will be seen, there is no doubt that the degeneration is in the early stage, proves, however, that this first case shows the commencement of the cord lesion of anæmia.

Case 1.—The patient was a man, aged 26, who was admitted into the Bristol General Hospital, under Dr. Harrison, for pneumonia, and died a few days later. He was profoundly anæmic; examination of the blood gave red cells 660,000 to c.m., irregular in size and shape; hemoglobin, 16 per cent.; blood decimal about $\frac{16}{13}$.

Besides the ordinary lesions of pneumonia and general anæmia of all the organs the chief changes found *post mortem* were as follows. The non-consolidated areas of the lungs were rusty in colour; the heart small, but not fatty; the pancreas somewhat indurated; the mucous membrane of the small intestines thin, and in parts atrophic; Peyer's patches inconspicuous; the intestinal mucosa nowhere gave a pink colour with mercuric chloride solution; no iron reaction in liver, spleen, or kidney.

The brain and spinal cord were apparently healthy, except that they were very anæmic.

Microscopic examination of the organs showed no noteworthy changes, except in the spinal cord. Sections were made of all parts of the cord, and exhibited partial degeneration of the posterior columns, entirely confined to the cervical portion, and not quite extending to the lower limit of that region.

The extent of the degeneration is shown in figs. 1 and 2. It was most extensive at the level of the third and fourth cervical roots, occupying the greater portion of the central parts of the posterior columns, not affecting, however, the anterior and posterior extremities of the column of Goll, and leaving an unaffected zone of varying width on the dorsal surface of the cord, and lining the posterior commissure and posterior cornua. At the level of the fifth and sixth cervical the degeneration formed on each side a narrow truncated area, the broad end dorsally, in the lateral border of the posterior median, and mesial part of the postero-external, columns. The posterior roots were normal, and there was no evidence of the entrance of degenerated fibres from them into the cord. Sections stained by Marchi's method showed a few scattered degenerated fibres in those parts of the posterior columns which, by the Pal-Weigert stain, appeared healthy, and also a very few in the lateral columns; both sets in the cervical region only.

The gray matter and other parts of the cord were normal.

Case 2.—Profound anæmia, three and a half years' duration; paræsthesiæ and paresis of legs after two years; recovery from anæmia; death from gangrene of lung following pneumonia; post mortem, stationary changes in cord.

J. S., aged 28, gardener. First admitted to hospital March 29, 1900. Family history good, both parents alive and well, and two brothers and three sisters are in good health.

He has never had any bad illness, nor suffered from anæmia previously. He has not had syphilis.

In 1898 he had a severe scalp wound which bled profusely, and about a year later he had profuse epistaxis from a blow from a stone.

For nine months he had been gradually losing strength, and had become anæmic. On exertion he had a sensation of tightness across the epigastrium, and pains in the back and legs. The feet and ankles became swollen towards night. For a week previous to admission he had been obliged to take to bed.

He was very pale in appearance; there was no jaundice. The pulse, 92, was soft and compressible, the tongue red and clean. Many of the teeth were carious.

The lungs were normal; systolic murmurs were audible at the base and apex of the heart, and over the great vessels. The lower edge of the liver could be felt, and the tip of the spleen came just below the costal margin.

The pulsations of the abdominal aorta were very plainly felt.

The urine was acid; Sp. gr. 1021; it contained a trace of albumen, no sugar, and showed a faint band of urobilin.

He was given iron and arsenic, and kept at rest in bed, and on this treatment he steadily improved. In June he left the hospital much better, but with still a trace of albumen in the urine, and tendency to œdema of the feet.

He returned and was re-admitted on April 25, 1901. He said that he had kept fairly well during the year, but had failed very much in the last six weeks. Since the beginning of the illness the slightest exertion had given him palpitation. Additional symptoms now were: twitching of the muscles of the legs at night, defective smell and taste, vomiting in the morning, a feeling of numbness in the fingers and toes, and loss of flesh. On examination he was well nourished; he was anæmic, but had not the lemon tint of pernicious anæmia; the pulse was 84, very compressible. The organs were in the same condition as previously. The tip of the spleen could just be felt. The knee-jerks and other reflexes were normal. The colon again contained scybala. The twitching of the leg muscles sometimes prevented sleep. The amount of urine passed daily was from twenty to forty ounces, it still contained a trace of albumin, and showed a moderately well-marked but diffuse urobilin band. Specific gravity 1025. There were no hæmorrhages in the fundus oculi, which was anæmic, but otherwise normal in both eyes.

From June 7 to 17 he was not so well, the temperature was raised to 100° with slight diurnal variations, and he was very weak, not being able to wash himself or get out of bed. There

FIG. 1.

FIG. 2.

FIG. 3.

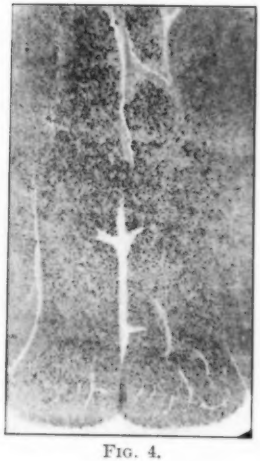


FIG. 4.

was also an increase of the urobilin in the urine, and the spleen became slightly larger.

From June 17 the condition of the blood gradually improved, he looked better, the spleen diminished in size, and the urobilin band practically disappeared from the urine. The skin was somewhat dark and pigmented. The muscles of the legs were weak and flabby, but not tender or distinctly wasted. Sensation was unaffected; the plantar reflexes were flexor in type, and feeble.

He now left the hospital, and was next admitted on April 3, 1902. He had kept in fair health until the beginning of the year, when he began to get very weak and to suffer from shortness of breath, palpitation, and giddiness.

He had a red flush on the cheeks, but the mucous membranes and the rest of the skin were pale. Over the chest, back, and limbs small round pigmented spots and petechiæ were fairly numerous. The pulse was regular, 76, full, and somewhat tense. The tongue was red and clean.

Physical examination showed that the condition of the organs was the same as on previous occasions, except that the liver was not felt. The spleen was slightly enlarged. The urine sometimes contained a faint trace of albumin. By Jaffé's test it appeared to contain a large quantity of indican, and with the spectroscope it showed a diffuse, but not intense or sharply-defined urobilin, band. There were also crystals of oxalates and phosphates.

During April and May his condition remained much the same; he was very weak and had no appetite. The temperature rose occasionally to 100° F. at night, falling a degree or so in the morning. He complained of numbness and tingling in the feet, especially in the right foot. Both optic discs and fundi were very pale, but there were no signs of past or present hæmorrhages. Towards the end of May he had a very bad attack of diarrhœa, after this he improved, and left the hospital at the end of June without any fresh symptoms. It should be added that at the beginning of June he was not so well, suffering much from pains in the back, abdomen, and limbs, and there was a rise of temperature to over 100° F. on several successive nights. The tingling and numbness in the legs and feet continued, but without objective loss of sensation, except that he had some difficulty in feeling a hot test-tube as hot. He was seen as an out-patient in October, and was then keeping well, though not very strong. His cheeks were red and his face generally a good colour; his chief complaint was of tingling and numbness in the feet and legs, and of some weakness. He attended as an out-patient

for about two months. He could walk a fair distance, and there was nothing remarkable about his gait. The knee-jerks were present, not very active; there was no wasting of muscles and no rigidity. Plantar reflexes weak and flexor in type. Very slight defect of sensibility to touch and prick over the lower extremities, especially on the feet.

The condition of the blood at this date showed very marked improvement.

He was not seen again until May 14, 1903, when he was brought up to the hospital in an almost collapsed condition. He was very drowsy, and at first refused to take any nourishment.

There were signs of consolidation of the lower lobe of the left lung, and the breath and sputum were horribly offensive. The pulse was 120, very feeble, and the heart's sounds almost inaudible.

The temperature was irregular, soon after admission it rose to 103°, and for some days afterwards varied between 100° and 104°. He was very dusky in colour. The abdomen was retracted, the spleen was not felt. The urine was retained, a specimen withdrawn by catheter was acid, Sp. gr. 1,030, with much albumin and many hyaline casts.

The lung symptoms steadily developed, and he died from heart failure ten days after admission.

During this last stay in hospital he was too ill to make a satisfactory examination of the nervous system possible; he came in with a sacral bed-sore (he was very ill at home for three weeks before admission), and one developed on the right heel shortly afterwards. So far as could be made out there was no decided loss of sensation to touch or prick over the lower extremities, no spastic contracture of them, and he could move them well. There was general wasting.

The following table gives the condition of the blood at intervals during the whole course of the illness:—

Chart of Blood.

	Red blood corpuscles.	Hæmoglobin.	Blood decimal.	White cells.	
March 27, 1900	2,800,000	50 per cent.	1	10,000	Slight increase in eosinophiles, in lymphocytes and mononuclear cells.
June 11, 1900	3,000,000	55 per cent.	$\frac{11}{12}$	7,000	
May, 1901	2,240,000	32 per cent.	$\frac{16}{25}$	8,000	Lymphocytes and mononuclears increased somewhat. Eosinophiles in normal proportion.
June 3, 1901	2,000,000	38 per cent.	$\frac{19}{25}$		
June 20, 1901	1,700,000	34 per cent.	1		
July 15, 1901	3,800,000	52 per cent.	$\frac{13}{10}$	5,000	

	Red blood corpuscles.	Hæmoglobin.	Blood decimal.	White cells.	
May 23, 1902	2,000,000	30 per cent.	$\frac{3}{4}$	4,000	Differential count showed normal proportion between kinds of white cells.
Oct. 16, 1902	3,420,000	48 per cent.	$\frac{13}{17}$	4,000	
May 15, 1903	4,100,000	80 per cent.	1	20,000	Specific gravity 1,061. A polymorphonuclear leucocytosis due to pneumonic changes.
May 20, 1903	4,100,000	75 per cent.	—	20,000	

With regard to the red cells, at the beginning of the illness, and at each subsequent relapse, there were numerous microcytes, macrocytes, and poikiloytes, with a small number of normoblasts. As the condition improved on each occasion, the red cells tended to return to the normal shape and size. No megaloblasts were seen at any stage. The *post-mortem* appearances were briefly: gangrene of lower lobe of left lung; general bronchitis; congestion of base of right lung; heart muscle pale and flabby, but not fatty; congestion of liver and spleen; kidneys intensely congested and indurated; marrow in ribs, sternum, and shaft of one tibia red in colour.

Sections of the various organs were stained with hæmatoxylin and eosin, hæmatoxylin and rubin and rosin, and osmic acid and carmine.

The following is a short summary of the microscopical changes:—

Heart.—No fatty change; increase of pigment round nuclei of muscle fibres.

Liver.—Congestion; cloudy swelling of liver cells, many of which showed vacuolation.

Spleen.—Intense injection; some excess of large pale cells in sinuses, apparently due to proliferation of cells lining them.

Kidney.—Chronic glomerular nephritis of moderate extent, affecting the convoluted tubes but slightly.

Marrow.—Excess of polymorphonuclear cells.

No iron reaction given by any organ.

The brain was normal.

Spinal cord.—By the Pal-Weigert stain the posterior columns showed a degeneration which, beginning in the upper dorsal region (see fig. 7) as a small area of degeneration in the posterior and central parts of Goll's column, extended throughout the cervical portion of the cord. In the cervical region the degeneration was almost entirely one of Goll's column, except that it extended a little on the dorsal surface of the cord to involve the posterior end of the postero-external columns (fig. 6). The

degenerated areas gave no reaction with Marchi's stain, most probably because the degeneration was of old standing, but in them were minute areas or cavities, from which the nerve fibres had disappeared. This same appearance was also noticed to a very small degree in the crossed pyramidal tracts for the extent of two or three segments in the upper dorsal region of cord only. The other parts of the cord were normal, as were also the gray matter, the anterior horn cells, and both anterior and posterior roots at all levels.

The interest of this case lies in the circumstance that the patient had recovered from his long continued anæmia, and died from an accidental lung affection. Further, the changes in the cord had not advanced very far, and apparently had ceased to extend; I think this is also the reason of the slight affection of the postero-external columns. The microscopic appearances and the clinical symptoms both point to the stationary character of the cord lesion. If this inference is correct, then the case illustrates the close connection of the cord changes with anæmia, and their dependence upon the latter. As showing the completeness of the recovery from anæmia the fact that the hæmato-poietic organs were able to cope with the strain of the fatal illness is important. Although in some respects, especially in the recurring episodes of blood destruction, the illness resembled pernicious anæmia, yet in most of its features, and notably in the fact of such complete recovery, it did not correspond with the latter condition. The state of the blood fairly corresponds to Cabot's third grade of secondary anæmia, the leucocytosis, which accompanies this form in the severer cases, not being very pronounced.

Case 3.—John C., aged 45, admitted under Dr. Harrison. The clinical notes of this case were very brief; it appears to have been entered in the hospital records as pernicious anæmia, but the notes, blood changes, and *post-mortem* appearances hardly bear out this diagnosis. The illness began nine months previously to admission with an attack of pneumonia, followed by severe anæmia, and for the last four months of this time he had had various paræsthesiæ, numbness and tingling in the hands and feet, with weakness of the limbs. There was no absolute paralysis, nor anæsthesia. He died of bronchitis, for which he came into the hospital, nine days after admission.

The blood examination gave 2,600,000 red cells; hæmoglobin 30 per cent.; blood decimal $\frac{3}{5}$. No increase of white cells. Red corpuscles varied in size; many poikilocytes; no megaloblasts or normoblasts.

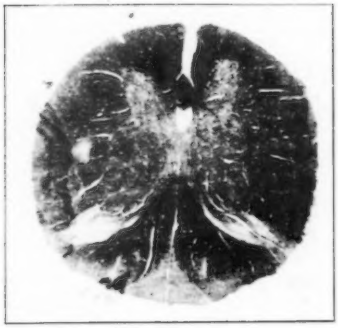


FIG. 5.

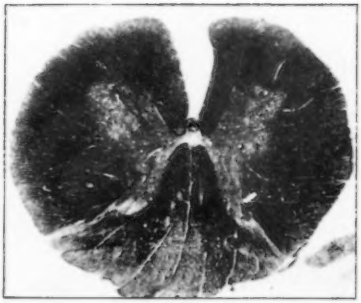


FIG. 6.

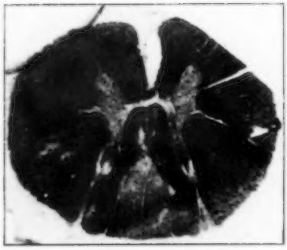


FIG. 7.

Post mortem.—The fat was bright yellow; some yellow transudate in serous cavities gave no urobilin band; general bronchitis; œdema of bases of lungs; no fatty changes present in the heart or other organs; kidneys very pale but not fatty; the liver and kidney gave a faint iron reaction, but not the spleen; the latter weighed 4 ozs., and appeared healthy; marrow healthy; small intestine showed patches of catarrh, no pink colour was obtained by treatment of mucous membrane with mercuric chloride solution; brain and cord healthy in appearance, except for the general anæmia, which affected every organ.

The above findings were confirmed by microscopical examination. The cord showed a more advanced degeneration of the posterior columns than in the two preceding cases, though here also the columns of Goll were most affected. The degeneration extended regularly, though slightly patchy in character, from the medulla to the level of the first lumbar roots, but not below this, being most intense in the cervical and upper dorsal regions. In the former the most intense degeneration occurred along the boundary between the postero-median and postero-external columns (fig. 8); it affected the greater part of these columns, leaving a marginal zone of varying extent, lining the posterior commissure and cornua and the dorsal surface of cord, free from disease. In the dorsal region the degeneration is rather less extensive, the marginal area being broader; the diseased part gradually diminished in passing down the cord until it became a very small patch on each side at the first lumbar segment, below which it could not be traced (figs. 10, 11).

In sections stained by Marchi's method a few scattered degenerated fibres were seen, as in case 1, in the lateral columns, (position of pyramidal tracts) and in the postero-external columns, in areas which by the Pal-Weigert stain appeared normal.

The degenerated fibres, as in the other cases, could be traced upwards to the posterior nuclei, chiefly nucleus gracilis, in the medulla, in which they ended (see figs. 3 & 4). The gray matter, posterior roots and other parts of the cord were healthy. For vessels, see general description.

Case 4.—This patient was one who died of anæmia in the hospital; but, unfortunately, I have no notes of the case, nor of the condition of the blood.

A complete examination was made of the spinal cord, brain, and some of the peripheral nerves. The brain and the nerves were healthy. In the cord degeneration of the posterior columns was found, extending through the cervical and dorsal regions, and

through the lumbar as low as the level of the fourth lumbar roots, where it ended, not involving the sacral region. The degeneration in this case is more extensive than in the foregoing cases (see figs. 12, 13, 14), and corresponds in this respect more to case 5. It was, however, as in the milder cases, most extensive in the cervical region and upper dorsal regions, in which the whole of the posterior columns were affected, except a very narrow bounding rim of healthy tissue. In the lower dorsal and lumbar regions this margin is larger.

In the lateral columns in the upper and mid-dorsal regions of the cord there was a small area of degeneration on each side (not shown in the figures) in the position of the pyramidal tracts, but this did not extend into the other parts of the cord.

Case 5 is published in full in the *British Medical Journal*, vol. ii., p. 325, with figures of the spinal cord degenerations. In this case the condition was apparently one of pernicious anæmia. She died nine days after admission to the hospital. The duration of illness was 12 months, during the *last four months* of which she had suffered from increasing weakness of the legs, with a feeling of deadness in them; when admitted there was some loss of motor power, no affection of sensation, and the plantar and deep reflexes were active.

The blood examination on admission gave red corpuscles 870,000, many poikilocytes, macrocytes, microcytes, and some normoblasts. White corpuscles 10,000, the increase being due almost entirely to polymorphonuclear cells. Hæmoglobin 30 per cent.; blood decimal ·6. Seven days later red cells 830,000; whites 30,000; hæmoglobin 25 per cent. The changes in the cord were most advanced in this case, as a reference to the figures will show; briefly they consisted in a fairly complete degeneration, though somewhat patchy in distribution, of the posterior columns extending the whole length of the cord, ending above in the nuclei of the posterior columns in the medulla, and in the lower dorsal and upper lumbar region of a small area of degeneration in the posterior part of the left lateral column.

This series of cases, I think, shows that the spinal degeneration which occurs as a secondary change in profound anæmia, and has been found and described by many observers, begins in the posterior columns and first in the cervical and upper dorsal regions of the cord. Further, that in cases which die from some cause in an early stage of the

cord disease, the affection may be unsuspected during life, and will be found limited to this region after death. In more advanced cases the posterior columns are involved almost throughout the cord, and the disease is then most extensive in the region in which it first begins, affecting here the whole of the posterior columns except a narrow rim of tissue on the dorsal surface and lining the posterior commissure and posterior horns: in the lumbar and sacral regions rarely spreading to so great an extent. These changes, though fairly regular, and possibly indicating that the fibres of the posterior columns which run the longest course are earliest degenerated, do not exactly map out special tracts, but are somewhat patchy in distribution, often varying at different levels for no obvious reason. The posterior roots do not seem to be affected. The appearances in some instances suggest some relation to the vessels, as the degeneration often seems to begin in the terminal distribution of the small artery that runs into the cord along the septum between the column of Goll and the postero-external column, that is to say, a small area about midway between the anterior and posterior ends of Goll's columns, involving slightly the postero-external columns. This relation is, however, though suggestive, not constant, and therefore no undue stress can be laid upon it.

In the cases in which the posterior column changes are most marked a patch of degeneration is also to be seen, either on one side or both, in the lateral columns in the position of the crossed pyramidal tract; this, again, is most evident in the cervico-dorsal or upper dorsal regions, but in one case appeared in the dorsi-lumbar region. It is more patchy and less regular even than the degeneration in the posterior columns, and does not extend over so great a length of the cord, in mild cases being limited to a few segments.[1]

It must be added to this that though the Pal-Weigert stain may indicate degeneration limited to the above situations, sections stained by the Marchi method often show a few

[1] A typical example of the affection of the pyramidal tract in these cases is figured in my paper in *Brit. Med. Journ.*, 1897. Vol. ii., p. 326, fig. 3.

scattered degenerated fibres occurring singly in a much wider distribution in the posterior, lateral, and occasionally a very few in the anterior, columns. Fibres stained black by this method also appear, if the case is a recent one, in the areas which by the Pal-Weigert stain appear to be already degenerated (see fig. 4). As regards the finer changes they may be said briefly to be practically identical with those described by Dr. Risien Russell in cases of "subacute combined degeneration" of the cord. The vacuolation resulting from the destruction and dropping out of nerve fibres is generally well seen.

In the degenerated areas there is an increase of the neuroglial framework, the vessels often show greatly thickened walls, and sometimes hyaline degeneration of them. In some of my sections the overgrowth of neuroglia seems to have been more extensive than was met with by Drs. Putnam and Taylor.[1] The older the patch the greater seems to be the sclerosis, the denser the structure of the part, and the less marked the vacuolated spaces, as if the tissues had contracted. In such cases the increased connective tissue may appear to spread directly from the vessel walls, and sometimes a leucocytal infiltration is seen around the latter. These changes seem to me to be entirely secondary to the parenchymatous degeneration of nerve fibres, and although, as alluded to above, this degeneration shows an apparent relation to vascular distribution, I should not attach further weight to this than that it may possibly indicate the route of conveyance of a toxin.

In favour of the secondary nature of the vascular changes is the fact that they are not evident in regions where the nerve degeneration is slight (and recent), and, further, are not present in areas of the cord where the only evidence of disease lies in the presence of a few scattered degenerated fibres. I found no patches of myelitis in my cases. The gray matter and its contained cells showed no important changes, the only noticeable one being the not infrequent presence in the cells of the anterior horn and of Clarke's

[1] "Diffuse Degeneration of the Spinal Cord." *J. Nerv. and Menta Dis.*, 1901.

Fig. 8.

Fig. 9.

Fig. 10.

Fig. 11.

column of an unusual amount of pigment. This pigmentation is also mentioned by Putnam and Taylor. The meninges were in all cases unaffected, and the peripheral nerves, where examined, normal.

To turn to the clinical features of these cases, clinical symptoms may certainly be absent, and the lesions in the cord not even visible at the *post mortem*, but only found after hardening and staining it. It is probable, therefore, that such degenerations in the cord are of more frequent occurrence in patients dying from anæmia than is generally supposed. As these changes are secondary and subsidiary to the general symptoms of severe anæmia, they may easily be overlooked, but my more recent experience is that a careful investigation will generally elicit some signs of the affection of the cord.

The most prominent clinical symptoms are certainly paræsthesiæ of various kinds, such as numbness, tingling, feelings of pricking, or of pins and needles, first appearing in the feet and legs. Later the hands and forearms may be affected, when a common complaint is, that numbness or deadness of the fingers renders fine movements difficult. These paræsthesiæ vary much in intensity during the course of the illness. Objective disturbance of sensation is not marked, but in the late stages may consist of some deficient appreciation of all forms of sensation over the lower extremities. After these sensory symptoms weakness in the legs may follow, often in the sense that they feel heavy, and later still both arms and legs may be weak. A frequent and often troublesome symptom, is twitching of the muscles, especially of the legs, and at night. The deep reflexes are present, ankle clonus is not obtained, and the plantar reflex, flexor in type, is sometimes difficult to get. The knee-jerk was absent towards the end of life in case 2, but he could only be examined as he lay in bed.

There is no ataxy, and no incontinence of urine.

In relation to the clinical symptoms, I should like to briefly mention another case, though the diagnosis was not confirmed by a pathological examination.

The patient was a middle-aged man who suffered from a

form of rhinitis, with a spongy condition of the nasal mucous membrane, which gave rise to recurrent attacks of profuse epistaxis. Under treatment in Hospital he was invariably relieved, but after an interval, generally of considerable duration, six months to two years, he relapsed, and the attacks of epistaxis returned. This epistaxis led to moderately severe anæmia of the chlorotic type, as regards the ratio of red blood cells to hæmoglobin. He was thus under observation for anæmia at intervals for over five years. The following is the chart of the blood, which indicates a progressive deterioration of it :—

	Red Cells.	Hæmoglobin.	White Cells.	
1896. Jan. ..	3,100,000..	31 per cent.		
(On leaving Hospital) March	4,300,000..	62 per cent. ..		
1899. Feb. ..	2,400,000..	25 per cent. ..	10,250	Many poikilocytes, normoblasts, small and large red corpuscles.
			Polymorphonuclears 69 per cent. Small lymphocytes 23 per cent. Large lymphocytes 4 per cent. Eosinophiles 4 per cent.	Poikilocytes, normoblasts, micro- and macrocytes numerous.
1900. July ..	2,750,000..	35 per cent. ..	9,000	As before.
			Differential count normal.	
1901. Feb. ..	2,400,000..	25 per cent. ..	12,000	
			Polymorphonuclears 67 per cent. Small lymphocytes 23 per cent. Large lymphocytes 8 per cent. Eosinophiles 2 per cent.	As before.

On the last occasion the anæmia only slightly improved under treatment, but on previous ones recovery was satisfactory, as in 1896, but I have not thought it necessary to give the actual blood counts each time.

Whilst in the Hospital in July, 1900, he complained for the first time of various paræsthesiæ, especially tingling and numbness in the feet and legs; and on his subsequent admission in 1901 these symptoms had very much increased, and were the source of great discomfort. He now com-

plained of deadness or numbness of the hands and fingers, varying from time to time, and sometimes interfering with the performance of fine movements. There was no decided objective loss of sensation, although it was perhaps somewhat dull over the feet. He also complained of some weakness in the legs, so that he could not walk so far as formerly. There was no ataxy and no muscular wasting; the knee-jerks were slightly exaggerated.

I have not been able to trace this man, but I have no doubt that the case was one of the same kind, *i.e.*, of a spinal lesion developing secondarily to repeated attacks of severe anæmia.

Hæmorrhage as the cause of anæmia in such cases deserves special mention, and is probably of importance; it occurred in two of my cases as the prominent etiological factor, and has been noticed in several others.[1]

The cases that I have described above form a fairly distinct group, and the changes in the cord occur in such a way as to show that they result from anæmia, and from forms of anæmia due to blood destruction. Though pernicious anæmia is one form which causes these cord lesions, it is by no means the only one. I am here taking pernicious anæmia in the ordinary sense of the term, though there is so much difference of opinion as to what actually constitutes pernicious anæmia that it is difficult to avoid confusion. In favour of the point that it is only when anæmia is due to blood destruction that these cord changes follow is the striking fact that they are not seen in chlorosis, of however long standing. A possible view of the pathology of these cases seems to me to be that of parenchymatous degeneration due to some toxin, possibly set free in the course of a pathological blood destruction, and therefore hæmolytic in origin. More extended observations on the blood, and on the precise character of the anæmia, would tend to elucidate the nature of the process.

In this particular group the cord changes often follow on so long a period of anæmia that it is difficult to suppose that

[1] *Vide* Dr. James Taylor, "A Case of Subacute Combined Sclerosis." BRAIN, 1904, p. 34.

the lesion is other than a secondary one, although it is possible that the cord changes might be due to the continued action of the same cause that has already produced anæmia, just as in those cases of cord degeneration and anæmia described by Dr. R. Russell and Dr. J. Taylor, they suppose that both conditions are due to the same toxin.

It may be mentioned, in passing, that in pernicious anæmia lesions in the spinal cord of quite another kind sometimes occur, namely, numerous small hæmorrhages, chiefly in the gray matter. Such cases are rare[1]; the hæmorrhages are to be compared with those found in the retina in this disease, and seem to me to have nothing to do with the cases now under consideration. There was no evidence in my sections that the degeneration started from any lesions of this kind.

The question now arises: Is there sufficient reason to put cases such as are considered in this paper, in which the cord changes distinctly follow or are secondary to profound anæmia of some duration, into a separate group, and to distinguish them from those in which similar lesions of the spinal cord occur, apart from anæmia; and further, what is their relation to the cases described by Risien Russell, Batten, and Collier[2] under the title of "Subacute Combined Sclerosis," and by Putnam and Taylor[3] and others, under the head of "Diffuse Degeneration of the Spinal Cord?" The latter writers say that "it seems to us too soon to make either clinical or pathological divisions in the general group of diffuse degenerations, and particularly to attempt the demonstrations of lesions peculiar to anæmia."

Such a division has been already attempted by Bastianelli in 1895, but his distinction, valuable as it is, is partly based on the acute or chronic course of the disease,[4] and will hardly hold good, for, as three of my cases show, and as other writers have also pointed out, the anæmic cases which, according to his classification, fall into the

[1] For a case of this sort see *British Medical Journal*, 1897, vol. ii., p. 326.
[2] BRAIN, 1890, Spring.
[3] *Journ. Nerv. and Ment. Dis.*, 1901.
[4] See J. Taylor, "A Case of Subacute Combined Sclerosis," BRAIN, Spring, 1904.

FIG. 12.

FIG. 13.

FIG. 14.

acute class, are, on the contrary, often chronic in course. I believe, however, he is correct in the view that, as a general rule, in the cases secondary to anæmia the cord changes are slighter, and most often limited to the posterior columns.

Without laying undue stress on the anatomical characters of these cases, it seems to me that there are good grounds, both clinical and pathological, for separating them from similar forms of degeneration of the cord, and that these are :—(1) The presence of decided or severe anæmia as the first, or sometimes the only, sign of illness ; preceding any evidence of spinal cord lesion. (2) Clinically symptoms and signs of disease of the cord are never very pronounced, and may be absent, though, possibly, in most cases are present when looked for. (3) Anatomically the lesion is generally confined to the posterior columns, and, if it affects the lateral columns, only does so to a small extent. And (4) it differs in extent and distribution from *subacute combined sclerosis* or *diffuse degeneration of the cord* in the following ways, according as the disease in the latter case is slight or severe. (*a*) In the milder cases of diffuse degeneration the degenerations are more precisely limited to the neuronic systems of the cords ; thus, in two cases under my own observation the columns of Goll were diseased from the lumbar region upwards to the medulla, and the crossed pyramidal and dorsal cerebellar tracts were similarly affected ; whilst in (*b*) the more severe cases, the degeneration is both much more extensive than in the cases considered in this paper, and also affects the periphery of the cord most, and appears to spread thence inwards, at any rate in the lateral and anterior columns.

On the other hand, it must be admitted that the microscopical details of the degeneration process appear to be similar in both groups, and further that there are cases which occupy an intermediate position, in which the cord changes are associated with profound anæmia, and are also very extensive in distribution. It seems to me that the chief difficulty lies in the nature of these intermediate cases.

In conclusion, although the evidence is not sufficient for a final classification, I think that a step in advance

might be made by separating into two distinct groups (1) the cases of pronounced anæmia with lesions in the spinal cord of the character described in this paper, and with slight or absent clinical signs of this disease; and (2) those of sub-acute combined degeneration or diffuse degeneration of the cord in which there is no anæmia, or, if it is present, it is late and consecutive to the appearance of symptoms of cord disease, and in which also the pathological changes in the cord are more extensive and their symptoms pronounced, following more or less closely the course outlined by Drs. Russell, Batten, and Collier.

In support of this division it may be mentioned that in fifty cases of the latter kind collected by Drs. Putnam and Taylor, in only seven was there decided anæmia. Those cases which seem to combine the features of the two groups, in presenting both profound anæmia and widespread cord degeneration, I should propose to leave provisionally in a class by themselves pending further investigation.

I am much indebted to Mr. James Taylor, of Clifton, for his kindness in preparing the photographs for me.

ILLUSTRATIONS.

Photo-micrographs of Cord.

CASE 1.

Fig. 1.—Cord at level of third cervical.
Fig. 2.—Cord at level of fourth and fifth cervical.

These figures show the point of commencement of degeneration as described in the text.

CASE 2.

Figs. 5, 6, 7.—Sections of cord at level of second cervical, third and fourth cervical, second dorsal.

(In section of second dorsal there is a light streak across dorsal free edge of left lateral and posterior columns due to irregularity in thickness of section.)

CASE 3.

Sections of cord at level of:—*Fig.* 8, third cervical. *Fig.* 9, seventh cervical. *Fig.* 10, seventh dorsal. *Fig.* 11, first lumbar: This photograph is too light in the anterior and lateral columns which were perfectly healthy.

Fig. 3.—Marchi stain. To show degenerated fibres ending in nucleus gracilis.

Fig. 4.—Marchi stain. To show about the number of fibres which stain black by Marchi in a degenerated column of Goll, which is also shown stained by Pal-Weigert's method in *Fig.* 8.

CASE 4.

Fig. 12.—Cord at level of eighth cervical.
Fig. 13.—Cord at level of tenth dorsal.
Fig. 14.—Cord at level of second lumbar.

A similar degeneration extended through the cervical region to the medulla.

SYSTEM LESIONS OF THE POSTERIOR COLUMNS IN GENERAL PARALYSIS, AND THEIR BEARING ON THE POINT OF ORIGIN OF TABES DORSALIS.

BY DAVID ORR, M.D.

AND

R. G. ROWS, M.D.

For the last three years, and more especially during the last year, while investigating the degenerations of the posterior columns in general paralytics, we have examined several cases in which the lesion was sufficiently pronounced to allow us to follow the course of the diseased fibres, and to justify us in coming to certain conclusions with regard to the origin of the earliest changes affecting the sensory spinal neurons of the first order.

We have found that the degeneration of these neurons has always begun at one definite spot in every case we have examined and in every segment of the various cords.

The reason why the starting point of these degenerations should be so constant will be pointed out in the course of the paper, and certain reasons will be advanced to show why the fibres should be vulnerable at this point. All the facts which we shall bring forward have been ascertained by an accurate examination of serial sections. Each cord has been examined from the lowest to the highest segment.

As we soon realised that no sclerosis was present in the majority of our cases, *i.e.*, that the lesions were in the early degenerative stage, all our tissues were submitted to treatment by the Marchi method, but care was taken to supplement this reaction with the Wolters and Van Gieson methods. The posterior roots were treated in exactly a similar manner.

The amount of lesion which we have found has naturally

varied, a fact which has been of the greatest assistance in the study of its exact starting point and in the localisation of the degenerated fibres in their first course in the cord.

Our results have been so constant, and the areas occupied by the degenerated fibres have so absolutely coincided with the sclerotic areas found in early cases of tabes dorsalis, that we have thought it worth while to record our results and conclusions.

We gave a preliminary account of these acute lesions at the meeting of the British Medical Association, held in Oxford last year (1), and since then we have completed the study of cases, in some of which sclerosis was beginning to replace the degeneration; in others the sclerosis was well established. We have, therefore, been able to follow the process in all its phases, from the slightest degree of degeneration onwards.

A study of the literature of tabes dorsalis shows clearly that there is considerable diversity of opinion with regard to the starting point of the process, but on one point all authorities are agreed, viz.: that the lesion is systemic and affects primarily the sensory fibres of exogenous origin. This fact at once paves the way for further research, because a lesion of this system is easily followed, on account of the accurate anatomical work which has been done since the introduction of the Golgi and Marchi methods. Bearing these two facts in mind, we can say that any lesion of this system which is of sufficient degree to enable us to map out its limits in the posterior columns, and to follow the degeneration of the terminals and collaterals of the fibres into the gray matter where they arborise around the nerve cells, is indistinguishable from a tabetic lesion in its course and distribution. It is of importance, therefore, that these early lesions should receive special attention, because their starting point is easily defined. On the other hand, we consider that the continued study of cases of tabes, in which sclerosis has replaced the degenerative phase, is of little assistance in enabling us to determine the pathogenesis of this disease.

Every one must admit that by the time a pathological

process has reached the stage of sclerosis many secondary factors have been introduced—which re-enforce the original exciting cause—and that an extension of the pathological process must have taken place in many directions.

The only accurate means by which we can determine the starting point of any disease, whether in the nervous system or elsewhere, is to first study it in the early degenerative phase. It is a neglect of this which accounts for the many theories which have been advanced regarding the pathology of tabes dorsalis.

Of the cases of general paralysis which we have examined it is only necessary to give one in detail, as the degenerations in the others affected the same system, and only differed in degree. The results obtained in this case by the Marchi method, and which may be seen in the accompanying photographs, were as follows:—

In all the sacral segments, the areas occupied by the sensory fibres of exogenous origin are thickly dotted with black droplets; scattered between these are many normal fibres.

At this level the exogenous fibres derived from the internal division of the posterior root are seen curving round the inner margin of the substantia gelatinosa Rolandi, and running forwards to occupy almost the whole breadth of the columns. They do not quite reach the commissure anteriorly nor the posterior periphery of the cord, and along both sides of the median septum a narrow strip is left practically free from degeneration. With the exception of these three areas, which constitute the endogenous systems, the whole column is well filled with degenerated fibres.

In the endogenous areas—whose limits will be shown later—there are very few degenerated fibres, so that these zones are sharply defined, and their limits are easily traced. The degeneration of the fibres of the exogenous system is seen to commence just at their point of entrance into the cord, and, in the extramedullary part of the root, no degeneration is present except in a small segment close to the cord.

Springing from the degenerated areas in the posterior columns, many fibres can be seen running into the gray matter. These first become evident in the fifth sacral segment, and proceed principally from the antero-lateral angle of the column, but also to a less extent from an area just anterior to Rolando's substance. In the first sacral segment these degenerated collaterals are seen

in greater numbers. A few traverse the internal corner of the substantia gelatinosa, but the great majority curve out into the posterior horn just anterior to this structure, and pass forwards to all regions of the anterior cornua. In the fourth sacral segment these collaterals could only be traced as far as the lateral group of cells in the anterior horns and, in the fifth sacral, the degeneration was confined to the posterior horn.

In Lissauer's area not a single fibre shows the Marchi reaction, and by the integrity of this region the rows of altered myelin in the internal division of the sensory root are thrown into bold relief. It might be stated here that not only in the sacral but in all other regions of the cord, Lissauer's tract shows practically no degeneration.

As mentioned above, the degenerated exogenous sensory system is sharply marked off from the endogenous. The anterior endogenous systems in the second sacral segment occupy the area just behind the posterior commissure, and extend for a short distance along both sides of the median septum, while the posterior systems occupy the postero-mesial parts of the columns and an oval area just anterior to this.

In the lumbar region the Marchi reaction of the lumbar root fibres is found scattered over the same areas as in the sacral segments; the degenerated exogenous fibres of the lower roots now occupy the regions by the median septum. Both the anterior and posterior endogenous systems pass lateralwards. Further, the degenerated fibres passing into the gray matter are more numerous, and can be seen streaming through the inner margin of Rolando's substance, and from a point just anterior to this. At the level of the second lumbar segment the degeneration reaches as far inwards as the median septum and as far forwards, in the middle line, as the posterior commissure. An area just behind the outer part of this commissure and extending for a short distance backwards immediately internal to the base of the posterior horn is found to be entirely free from degeneration with the exception of a few isolated fibres. This is the cornu-commissural layer of endogenous fibres now occupying a more lateral position than in the sacral region.

Just behind the endogenous tract at this level of the cord, a well-marked bundle of degenerated fibres makes its appearance. This arises from the cuneate fasciculus and passes into the gray matter to arborise around Clarke's nucleus. The terminal arborisations of these fibres amongst the cells of the nucleus are well shown by the Marchi method. With the displacement of the

posterior endogenous system outwards, the degenerated exogenous system extends further inwards and more towards the periphery of the cord, so that the area formerly occupied by Flechsig's oval is now filled with degenerated fibres.

In the first lumbar segment the degeneration in and around Clarke's nucleus, and of the fibres passing forwards to terminate around the cells in the anterior cornua is especially well marked.

From this point upwards there is a steady decrease in the degeneration affecting the intramedullary part of the roots and middle root entry zones, with a consequent diminution in the number of degenerated fibres passing into the gray matter.

At the level of the tenth dorsal segment the degeneration in the root entry zone is distinctly less, but more marked on the left side, and does not extend so far internally and posteriorly as in the lumbar region. A triangular area around the hinder portion of the median septum is thus left whose base is formed by the periphery of the cord, and in which the preponderance of normal fibres is much greater than in the entry zones, fig. 4. The separation of the degeneration in the most internal part of the posterior columns from that in the entry zones is more distinctly seen a little further up the cord, and in the sixth dorsal segment we find a very well defined area almost entirely free from Marchi reaction between these two regions.

In the anterior part of Goll's column the degenerated fibres are collected around the median septum, but in the posterior part they are scattered somewhat diffusely.

In the second dorsal segment there is still less degeneration in the entry zone which is more evident, however, on the left side. In Clarke's column and in the anterior cornua, the degeneration is very slight. The anterior and internal parts of the cuneate fasciculus—just behind the posterior commissure—contain a very considerable number of degenerated fibres, while in Goll's column the large majority of the degenerated fibres lie along the posterior third of the median septum and spread outwards from it for a little distance at the periphery of the cord. In the first dorsal segment the degeneration in the posterior columns, with the exception of that in Goll's tract, is again reduced. In Clarke's column of cells there is only a trace of Marchi reaction.

On passing from this segment up into the eighth cervical we find a return of degeneration in the entry zone and in the intramedullary part of the root. This can be traced back as far as the point of entrance of the fibres into the cord, but not beyond. The degeneration is more marked on the left side, and many degenerated fibres can be traced into the posterior and anterior horns.

In the seventh cervical segment the degeneration is seen to pass inwards, and the degenerated fibres are found scattered in the anterior part of the cuneate fasciculus on both sides as far forwards as the posterior commissure; and along the inner margin of the cuneate fasciculus where it lies adjacent to the column of Goll the fibres form a distinct band which, however, is more pronounced on the left side. Degeneration in the gray matter is still present. There is a slight increase of degeneration in the right root at this level.

In the sixth cervical segment the degeneration in both entry zones is diminished, and the band of degeneration along the inner margin of the cuneate fasciculus is not quite so well defined on the right side. On the left side there is a very distinct layer with its broad end directed towards the commissure and its tail pointing backwards. In Goll's column the degenerated fibres form an L-shaped figure on both sides of the postero-median septum. The degeneration in the gray matter is much diminished. A little further up the cord the degeneration in the entry zones disappears, and the layer along the inner margin of the cuneate fasciculus diminishes, while in the anterior part of Goll's tract at the level of the second cervical segment there is a sensible increase in the number of degenerated fibres.

In the posterior roots—outside the cord—treated by Marchi's, van Gieson's, and Wolters' methods, there is not sufficient change to account for the intramedullary lesion. Marchi's method shows a few scattered droplets while the two latter methods yield almost entirely negative results in both cord and roots.

To briefly recapitulate the points of importance in the case, we find:—

(1) Degeneration of the internal division of the sensory roots in their intramedullary path, commencing at the point of entrance into the cord. The maximum of degeneration in this system is found in the lumbar region, and it becomes progressively less in the dorsal cord from below upwards. At the eighth cervical segment there again occurs a slight increase, as shown by the Marchi reaction in the intramedullary part of the root, and by the degenerated layer along the inner margin of the cuneate fasciculus in the higher cervical segments.

(2) Complete integrity of the external division of the sensory root which consists of fibres of small calibre, and, in the cord, forms Lissauer's tract.

(3) The collaterals and terminals of the sensory system which pass into the gray matter, share in the degenerative process, viz.,

those passing into the posterior and anterior horns, and those arborising around Clarke's nucleus.

(4) The long fibres derived from segments in the lower parts of the cord are affected, as shown by the position of the degeneration in Goll's tract in the cervical region. This degeneration is small in amount, owing to the fact that under normal circumstances only a few fibres of the lower roots ever reach the higher segments of the cord, the great majority terminating around cells in the gray matter at the various cord levels, and also because not all the fibres in the internal division of the roots of this case were attacked.

Taking into consideration the integrity of Lissauer's area and the absence of degeneration in the extramedullary part of the roots, it is obvious, therefore, that the internal division of the sensory fibres has been attacked at its point of entrance into the cord, and that in consequence of this injury an upward degeneration has taken place, reaching as far as the terminals and end arborisations.

In all the cases besides the above, examined up to the present time, we have noticed a striking similarity with regard to the point at which the degeneration of the intramedullary portion of the posterior root fibres has begun, and in the distribution of these fibres in their course up the cord. Whenever a degeneration has been found it has always begun at the point where the fibres have entered the cord, and has been traced along a definite course.

We shall be better able to understand the distribution of the lesions if we briefly consider the path of the peripheral sensory neurons in the cord.

The posterior columns contain two sets of fibres, and each of these has a distinct origin and a definite course in the cord. One set is derived from the cells of the posterior root ganglia, and reaches the cord through the posterior roots. These are the exogenous fibres. The other takes its origin from the cells of the gray matter of the cord, and is, therefore, described as endogenous.

The septo-cornu-commissural endogenous system occupies a different position in the various regions of the cord. In the lower sacral region it is septo-commissural, in the lumbar region it is cornu-commissural, and in the higher

portions of the cord it spreads further back along the inner margin of the posterior horn.

The posterior endogenous system in the lower part of the cord is represented by the oval field of Flechsig and the triangle of Gombault and Philippe.

The embryological researches of Flechsig and Trepinski have led them to describe four zones in the posterior columns, which are determined by the period at which the fibres receive their myelin sheath. These zones, however, contain both exogenous and endogenous fibres, and are known as the posterior root zone, which consists of a lateral and a median portion—the anterior root zone, the middle root zone, and the median root zone.

Homén (2) and Philippe (3), in their articles on tabes, state that this division of Flechsig is not sufficient to indicate the course of the exogenous fibres in the posterior columns which are affected in this disease, and they suggest that it is necessary to follow the anatomical distribution of the exogenous system.

The first zone which the root fibres occupy after they have entered the cord is spoken of by Homén as the "root-entry zone" "Würzeleintrittszone" (fig. 1A). This root-entry zone lies along the inner and hinder portion of the posterior cornu. Medialwards it extends, more or less, into the column of Burdach. In the lower segments of the cord it is somewhat oval in shape, but in the upper parts it is narrower and elongated sagitally.

As the fibres ascend the cord they pass inwards and forwards along the inner margin of the posterior cornu, being pushed in these directions by the fibres of the succeeding roots.

From this zone many fibres, terminals and collaterals, stream into the gray matter of the posterior horns. The fibres which enter from the posterior part of the root-entry zone pass through the tip, and those from the anterior part through the inner margin of the cornu.

Some of these fibres arborise around the cells of the gray matter of the posterior horn; others pass forwards to the cells of the intermedio-lateral region and the anterior cornu, and many go to Clarke's nucleus.

The longest fibres of the posterior roots then form a long, narrow band, curved with its concavity outwards, which extends from the cornu-commissural angle in the anterior root zone, backwards through Burdach's column to the periphery of the cord. The fibres of this band continue to be pushed inwards and backwards by the successive roots which enter the cord, and the longest fibres of the posterior roots finally occupy the positions indicated in the accompanying diagram (fig. 1A).

Now, when we turn to the sections of the various cords which we have examined, we find that the areas occupied by the degenerated fibres correspond exactly with those which have been mentioned above in describing the intramedullary path of the posterior sensory neurons. Moreover, we are able to trace the course of the short and long fibres, terminals and collaterals in these areas by the Marchi reaction.

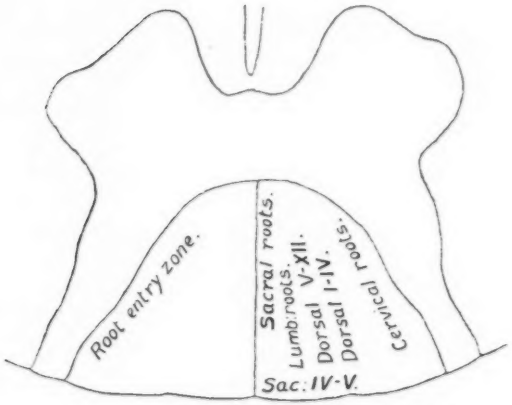
The degeneration of the fibres in these cases has invariably begun at the point where they dip into the cord.

Thus, in the case detailed, in the sacral and lower lumbar segments much degeneration is present in the intramedullary portion of the posterior root fibres, the root-entry zone of Homén.

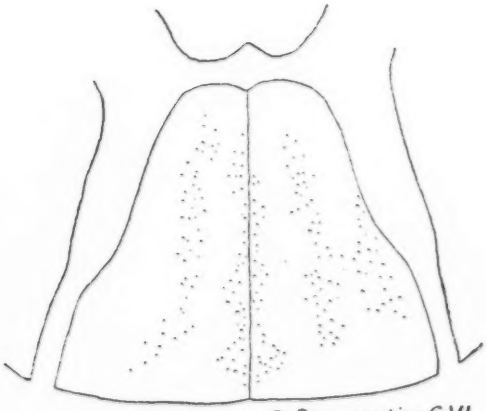
We would state here that by the term root-entry zone we wish to indicate the area outlined in the diagram (fig. 1A).

In the second sacral segment (fig. 2) it will be seen that there are numerous affected fibres in the root-entry zone, around the angle of the posterior horn and along its inner margin. But the area behind the posterior commissure and inside the neck of the posterior horn on both sides, together with the areas on each side of the median septum and along the periphery of the cord, contain very few degenerated fibres. These are the endogenous areas of the second sacral segment.

In the second lumbar segment (fig. 3) the root-entry zones are still thickly studded with degenerated fibres, as also are the areas along the anterior half of the median septum. These are the long fibres of the roots which have entered lower down and have been pushed inwards.



A (after Edinger)



B. Degeneration C. VI
Marchi method

FIG 1.

Drawn by Dr. Le Fann.

The anterior endogenous system is here cornu-commissural in position, *i.e.*, it has passed outwards, and now occupies the area inside the cornu-commissural angle and the inner margin of the posterior horn for some distance backwards. This area is easily distinguished by the absence of degenerated fibres.

The posterior zone, again, contains only a few scattered droplets.

As we pass further up the cord the posterior columns contain fewer degenerated fibres. This is due to the fact that there is less degeneration in the intramedullary roots, and also that fibres are continually passing out of the columns into the gray matter.

It is obvious, therefore, that the degenerated fibres situated in the parts internal and posterior to the entry zone are fibres of longer course, which have entered by lower roots, and some of which will reach the highest levels of the cord in Goll's tract.

But although there is a progressive diminution in the amount of degeneration as we ascend the dorsal region, we find that in certain roots there is a definite increase, which gives rise to a corresponding increase of degeneration in the root-entry zone. This allows us to follow more clearly the course of the fibres in the first few segments after they have entered the cord. For example, the left eleventh dorsal root contained more affected fibres than the tenth or ninth, and the fibres of this root can be traced inwards and forwards along the inner margin of the posterior horn in the next two or three segments.

Again, the intramedullary portion of the right eighth dorsal root fibres (fig. 4) also showed more degeneration than the adjacent roots. These fibres could be followed inwards and forwards as they ascended the cord, and at the sixth dorsal they reached to the commissural angle.

These variations in the amount of degeneration of the intramedullary portion of the sensory neurons are important, and explain the curious changes in the distribution and amount of degeneration which may be present in any segment of the cord, and which sometimes makes it difficult to understand the systemic character of the lesion.

FIG. 2.
Second sacral. *Photomicrograph.*

FIG. 3.
Second lumbar. *Photomicrograph.*

It will be remembered that on reaching the upper limit of the dorsal region the lesions in the posterior columns were less marked than in any other portion of the cord.

Immediately we examine the lower cervical region we find that there is again an increase of degeneration in the intramedullary portion of the root fibres, and in the root-entry zones. This gradually passes inwards and forwards, and ultimately forms a definite band along the inner margin of Burdach's column (fig. 1B).

In Goll's column the degenerated fibres, which are derived from the lumbar and sacral roots, lie in its internal and posterior portion.

Between this zone and the degeneration in the internal part of Burdach's column, there is an area in which very few altered fibres are found. This is the area normally occupied by the dorsal roots 12-5 [Edinger (4),] (fig. 1A & B). The absence of degenerated fibres in this area is explained by the fact that the intramedullary portion of the fibres of the dorsal roots were much less affected than those below.

We must now turn again to the various segments to consider the course of the terminals and collaterals which enter the gray matter, as they are demonstrated by the Marchi reaction in our cases.

In the second lumbar segment (fig. 5) numerous degenerated fibres, terminals and collaterals, may be seen passing from the root-entry zone, some through the substance of Rolando, and forwards through the gray matter; others through the inner border of the posterior horn, and then forwards for various distances.

This picture is not confined to any particular segment, but is invariably seen in every segment in which there is much degeneration present in the root-entry zone.

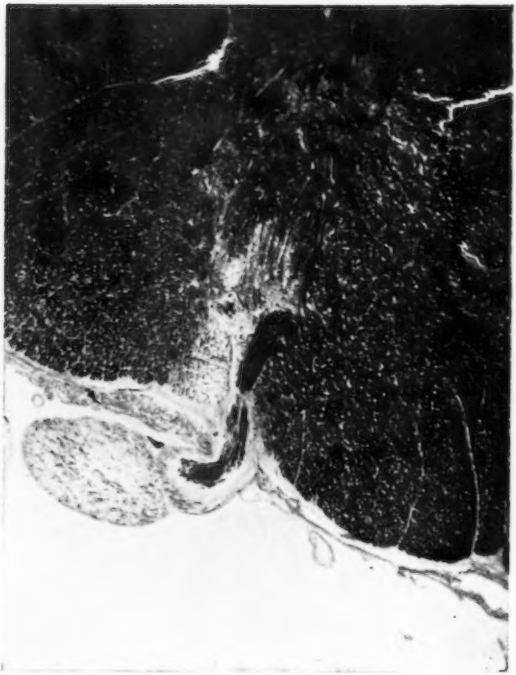
By referring to the photographs which are published with this paper, it will be possible to follow the course of these degenerated fibres in the gray matter. Most of those which pass into and through the substance of Rolando end by arborising amongst the cells of the posterior horn.

Of those which enter through the inner margin of the posterior horn, some end in the adjacent gray matter, but many have a longer course.

The fibres which enter at the angle of the posterior horn pass outwards and forwards towards the intermediolateral region; others forwards to the anterior cornua. But the most noticeable are the numerous affected fibres which pass into Clarke's nucleus. These are well seen in fig. 6, which is a photograph of Clarke's column in the first lumbar segment.

We have now followed, by Marchi's method, the course of the intramedullary portion of the exogenous fibres from the point at which they have entered the cord to their termination, and we find that the course of the fibres in the posterior columns, and of the terminals and collaterals in the gray matter, corresponds absolutely with the paths of these fibres as they have been described in the most recent anatomical works (Ramon y Cajal, Golgi, Edinger, Lenhossék, van Gehuchten, Homén, and others). We have pointed out also the areas which the endogenous fibres occupy in the posterior columns, and we have shown that these areas contain few degenerated fibres in all our cases. Moreover, the tract of small fibres known as Lissauer's tract, which is the external portion of the posterior root, has in no case shown any appreciable number of degenerated fibres (fig. 7).

There remains one other anatomical point to be considered. We have seen that in all the segments of these cases the degeneration of these root fibres has always commenced at the point where they have entered the central nervous system, and the question arises, is there any anatomical condition here which will assist in the explanation of this constant appearance? Obersteiner (5), in his "Anatomy of the Central Nervous System," describes the histological structure of the posterior root, where it enters the cord, as follows: "At the point where the posterior root pierces the pia mater it is constricted, and sometimes to a marked extent." "This reduction in size takes place entirely at the expense of the myelin sheath. Consequently, when stained by Weigert's method, the root at this point remains colourless, and a bright band, usually convex on the outer side, is seen traversing the root at the point of constriction."



Photomicrograph. Sanger-Shepherd three-colour process.

Face p. 472.

Having noticed the regularity with which the Marchi reaction has begun on the central side of this constriction, only a few droplets having been seen on the distal side, and these immediately near the ring, we have examined this region in many posterior roots by the Kultschitzky-Wolters and the van Gieson methods in serial section. (See coloured plate.)[1]

The former method shows the blue-black myelin sheath of the intramedullary portion of the posterior root fibres separated from that of the fibres in the root itself by a clear zone, in which no stain is visible.

By the van Gieson method, which stains the myelin yellow, and the axis cylinder and the fibrous structures red, we find that at the position of the clear ring mentioned above, the yellow colouration with the picric acid is absent, and, instead, this zone is stained a deep red. We also see that the deep red of the acid fuchsin is much more marked in the root outside the cord than it is in the intramedullary portion, while with the yellow stain of the picric acid the reverse is the case. These appearances depend on the histological structure of the root fibres outside and inside the cord. The fibres, in their extramedullary portion possess a sheath, the neurilemma, outside the myelin sheath, which is wanting on the fibres in their intramedullary course; and the myelin sheath itself is much thinner around the fibres on the ganglion side of the ring. At the ring of Obersteiner the neurilemma is supposed to blend with the pia mater.

We have been doubtful sometimes whether the myelin sheath is entirely absent at this point, but, with the methods at present at our disposal, its absence seems to be probable.

At this point, therefore, there is a change in histological structure, which consists of a loss of the neurilemma, and an absence, in all probability, of the myelin sheath. The appearances produced by the Marchi method support this view, because no black reaction has been found at this point.

[1] We have to acknowledge the assistance of Dr. Bythel (Manchester) in the preparation of this photograph.

The anatomical distribution of the degeneration shows us that we have here to deal with a system lesion of the sensory neurons of the first order, which springs from a definite point in every instance, and affects, to a varying extent, the main trunks and branches of the posterior roots in their intramedullary course.

After repeated examinations of the posterior roots, between the ganglia and the cord, we can state that no appreciable degeneration or other morbid change has been discovered by the methods of Marchi, Wolters, or van Gieson. It is true that by Marchi's method a few fine isolated drops have been seen, but it is impossible to associate such an appearance with the mass of degeneration in the cord, in the relation of cause and effect. No matter whether the amount of the degeneration in the cord be great or small, nothing more than a few isolated droplets has been found in the roots. (This statement holds good for the anterior roots also.)

It might be objected that the degeneration of the roots had been present at some former period and had undergone absorption, but the coincidence would be indeed unique if the absorption of the altered myelin had advanced to the same point in all the affected segments in our cases, as well as in those of other observers. We must remember also that the age of the degeneration varied in the different cases, and even in the different segments of these cords. Further, the methods of Wolters and van Gieson showed neither atrophic nor sclerotic changes, which they certainly would have done if degeneration had been present.

The absence of the degeneration in the roots makes it at once apparent, therefore, that the intramedullary lesion is not secondary to destruction of the ganglion cell or to disease of the root itself, and we are left with the only other alternative, that the sensory fibres have been attacked somewhere in their intramedullary portion, and we maintain that it is at the point where they enter the cord that they have been attacked by some noxious agent, which has been sufficiently strong to produce a degeneration which has then spread in a centrifugal direction. This view is

FIG. 4.
Eighth dorsal. *Photomicrograph.*

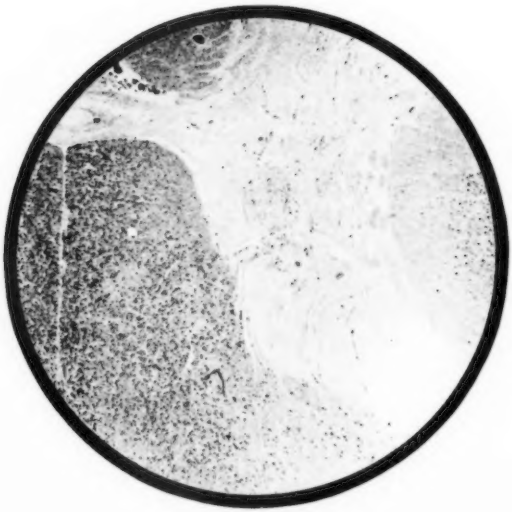


FIG. 5.
Second lumbar. *Photomicrograph.*

directly opposed to that of Nagoette (6), but to this point we shall refer later.

That noxious agents can bring about the degeneration of nerve fibres in any part of their course has been well established, both by experimental and pathological work, and we need not here enter into a detailed account of these primary degenerations of the cord. They have been repeatedly produced experimentally, and have been constantly found in the various anæmias, cachexias, intoxications, and infections, *e.g.*, chronic uræmia, diabetes, tumours. In short, they may be caused by the action of any poison on the nerve fibre, or by a disturbance of its nutrition. The condition is characterised by a progressive atrophy of the myelin sheath, while the integrity of the axis-cylinder is maintained for some considerable time.

But a primary degeneration may pass on to become a secondary one if the action of the poison be prolonged and the axis-cylinder attacked, so that, given a toxic agent in contact with a nerve, there is always a risk of secondary degeneration. And, according to Lugaro (7), the stronger or more localised the action of the toxin, the more often does secondary degeneration take place, whereas a diffuse and weaker toxic agent tends to produce a purely primary change.

Lesions very similar in distribution to those in our cases have been observed by Bruce (8), Homén (9), Nageotte (6), Williamson (10), and Erbslöh (11).

The degenerations of the posterior columns in general paralysis and in cases of cerebral tumour, described by Homén (12), coincide in origin and distribution with those which we have described. He has found that the degenerative process begins just inside the margin of the cord, and affects the exogenous zones principally and the collaterals, especially those concerned in reflex action. The lesion is more marked in the cervical and lumbar legions.

Another observation which he has made, and which we have been able to confirm, is that the changes in the posterior columns of the higher segments are in direct continuity with the changes in the lower part of the cord, after making due allowance for the passage of degenerated fibres into the gray

matter, and for the influx of fresh degeneration at intermediate levels. This point we have emphasised above, and we have also drawn attention to the modification in form which the degeneration in the entry zone exhibits when the fibres which have entered at any single root level are more affected than are those which have entered in the adjacent segments.

Erbslöh (11) also has stated that it is shown by the Marchi method, that the degeneration begins where the neurilemma is lost, and that, so long as the fibres in the root are covered by the neurilemma, they show no degeneration. He also affirms that at the ring of Obersteiner-Redlich, both the axis-cylinder and the myelin sheath become disintegrated.

The recent observations of Nageotte (6) on the changes in the posterior columns in cases of cerebral tumour are very interesting. He finds that the Marchi method begins to give a positive result immediately the sensory fibres enter the cord. The exogenous system is affected, including the collaterals and the fibres passing to Clarke's column. Lissauer's area shows no Marchi reaction, nor does the extramedullary portion of the posterior and anterior roots, but a definite inflammatory focus exists at a point just central to the root ganglion.

This inflammatory focus we have never seen, but, with the exception of this, the lesion which he has described is similar to that which we have found in our cases.

According to Nageotte, the neuritis produces a Wallerian degeneration which begins at the distal extremity of the fibres and spreads backwards towards the trophic centre. In his cases the process is assumed to have reached only to the point where the fibres enter the cord.

This assumption of Nageotte is based upon his acceptance of the opinion of Ranvier, that the Wallerian process travels centripetally along the fibres, *i.e.*, from their terminals towards the point of injury. But there is strong evidence against this view in the work of Erb, Tizzoni, and Neumann (13), and in the researches of Mönkeberg and Bethe (14). These last two authors, working with the tissues of cold-blooded animals, have shown that in the peripheral portion of a divided nerve the degenerative process always proceeds

FIG. 6.
First lumbar. *Photomicrograph.*

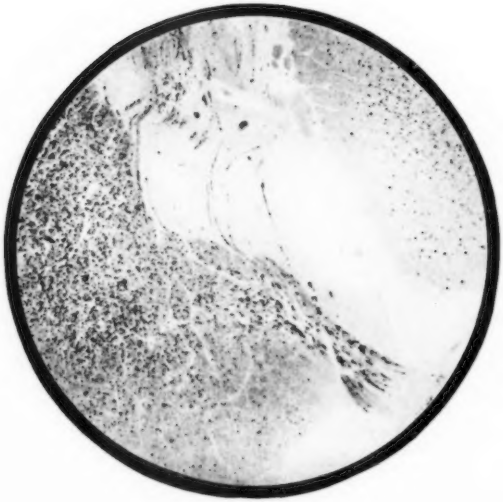


FIG. 7.
Second lumbar. *Photomicrograph.*

centrifugally from the point of injury in both the axis-cylinder and in the myelin sheath.

Moreover, we have often seen some degeneration of the fibres immediately after their entrance into the cord and in the root-entry zones, while little or no change was discernible in the collaterals in the gray matter. If Nageotte's view were correct, cases would be met with in which the Marchi reaction would appear first in the gray matter, and the degeneration would spread backwards into the entry zones, and the portion of the fibres near the margin of the cord would be left intact. As far as we can discover this has not yet been observed, and Nageotte has still to explain why the degeneration invariably reaches the Obersteiner-Redlich ring and stops there.

The integrity of Lissauer's area he attempts to explain by suggesting that the fibres of this tract are of endogenous origin, and, therefore, are not influenced by the neuritic process. Although we are in agreement with Nageotte with regard to the distribution of the intramedullary lesion, yet, in view of his histological and pathological facts, we cannot accept his explanation of the integrity of the extramedullary part of the roots and of Lissauer's zone. Why the latter should escape in acute lesions is certainly difficult to explain, but before we can accept that Lissauer's tract is composed of endogenous fibres we shall require a much stronger argument than that advanced by Nageotte. The mere fact that it does not participate in the degeneration is no proof of its endogenous origin, and we must bear in mind that the fine fibres in the peripheral nerve, which go to form this tract, have been shown by Ballance and Stewart (15) to degenerate more slowly than the coarse fibres after division of the nerve. This suggests that they possess a greater power of resistance. In view, then, of the weight of evidence which supports the theory that the secondary degeneration proceeds from the point of injury in a centrifugal direction, we regard the point at which the sensory fibres become part of the central nervous system as the focus at which these acute lesions of the posterior columns commence. We are convinced that such lesions are not the result of injury to the ganglion cells

or roots, and we have not been able to discover any changes in the meninges at the Obersteiner-Redlich ring, which could act mechanically on the fibres. But, from our investigations, we have arrived at the conclusion that these degenerations are caused by some noxious agent which circulates in the lymph and acts on the nerves at the point where they lose their neurilemma sheath.

That this point in the path of the sensory protoneurons may be regarded as one of special vulnerability is shown by the study of the acute and chronic system lesions, as well as by the recent work done to determine the direction of the flow of the lymph stream in the spinal roots and in the posterior columns of the cord.

Our knowledge of the course of the lymph stream in these regions has been added to considerably during the last few years. According to d'Abundo (16), and Guillain (17), the lymph in the posterior columns pursues an ascending course, and, at the same time, passes forwards towards the central canal.

That the lymph spaces of the pia covering the posterior part of the cord communicate with those of the posterior columns is shown by the diffusion of coloured fluids (Guillain). In their last work, Marie and Guillain (18), assert that there is a common lymph system—distinct from the other regions of the cord—for the posterior roots, the posterior columns, and the pia covering them, and that the lymph is not contained in vessels, but flows freely in the intervals between the fibres and the other structures.

Homén's experiments (19) show that the lymph in the peripheral nerves ascends to the cord, and that toxins are transmitted more readily by the posterior than by the anterior roots, so that the posterior columns are more exposed to irritants than the other parts of the cord.

This upward passage of the toxins in the sheath of the spinal roots we have ourselves observed in a case of staphylococcic infection of the tissues around the spinal ganglia of the cervical region (20). From this focus the toxins spread up the roots without injuring them, but at the point where the fibres entered the cord the Marchi method gave a

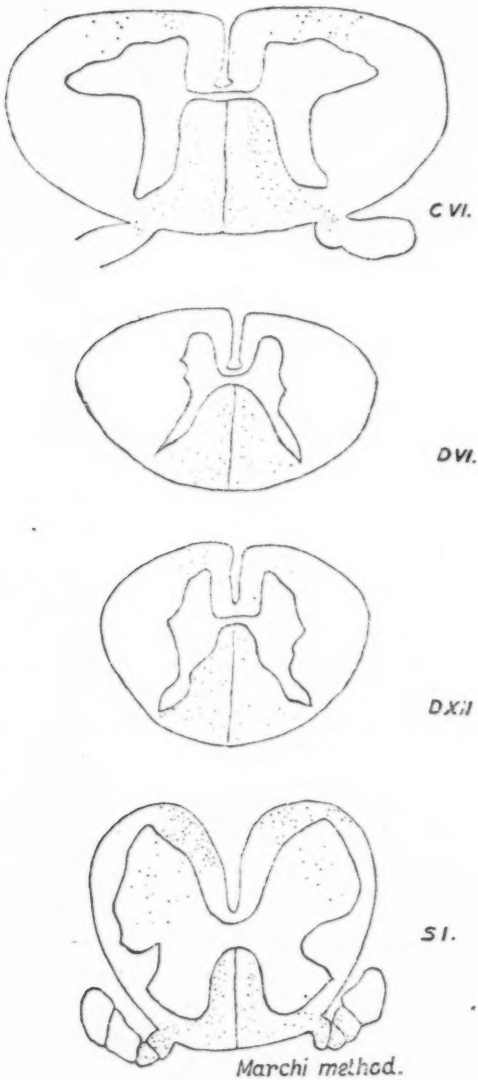


FIG. 8.

Drawn by Dr. Le Fann.

positive reaction, which was continued into the column of Burdach.

It is possible that the existence of an ascending stream of lymph from the large nerve trunks into the posterior columns may help to explain why some degenerative lesions of the sensory fibres attain their maximum in the cervical and lumbar regions. One of our cases certainly tends to support this result, because, with the existence of bilateral bedsores, that on the left buttock being the more severe, we found that there was more degeneration in the corresponding left posterior column in the lumbar enlargement; while, with a similar lesion of the right elbow, there were many more degenerated fibres in Burdach's column on the right side in the cervical region (fig. 8).

But in cases of diabetes, cerebral tumour, general paralysis, &c., in which no peripheral toxin producing focus exists, and yet the degeneration of the entry zone is more marked at the enlargements, some other factors must come into play in the determination of this peculiar distribution.

We are, however, certain of this fact, that whether the cerebro-spinal lymph is toxic from a peripheral source or from a general intoxication, the sensory fibres are prone to give way at the point where they lose their neurilemma sheath.

Speaking generally, the dorsal nerves are less affected than the cervical or lumbo-sacral nerves, and, moreover, the number of degenerated fibres in adjacent roots may differ considerably, and thus, as we have stated before, the systemic character of the lesions may appear to be modified at different levels.

The initial change in the injured nerves is a primary degeneration, but owing to the intensity of the poison and its continued action at the vulnerable point the axis-cylinder is early attacked and a secondary degeneration rapidly ensues. At this stage the process, spreading centrifugally along the nerves, can be designated by the term secondary degeneration, because the implication of the axis-cylinder cuts the intramedullary part of the fibre off from its trophic centre.

The first portion of nerve left uncovered by the neurilemma suffers initially, but with the toxic lymph passing upwards and forwards the neurons are probably injured at other points in their course, *e.g.*, in the entry zone. To take an example from the case, which is given in detail, it is evident that the fibres of any given lumbar segment must have come in contact with toxic lymph not only in the segment to which they belonged, but also with the lymph entering the segment immediately above.

It is obvious, therefore, how this degeneration differs from the Wallerian change, which is seen in the peripheral portion of a divided nerve.

But the process is different only in development; the end is the same: a fact which is proved by the involvement of the collaterals and terminals in the gray matter, and by the degeneration of the long fibres in Goll's column. Here one sees only the centrifugal extension of the secondary degeneration.

Now, given a secondary degeneration of a system of fibres in the central nervous system there never occurs a regeneration by means of which new nerves are formed. Complete regeneration is said to occur in the cord of the lower forms of animal life, *e.g.*, in amphibia; but Spiller and Frazier (21) have never found the slightest trace of it in dogs. Gradual absorption of the degenerative products, with progressive sclerosis, must be the end of these system lesions.

This is frequently found in the cords of general paralytics in all stages of development.

A sclerosis in the posterior columns is exactly what one finds in early cases of tabes dorsalis, so that the degenerations under consideration acquire more importance when we apply them to the study of this disease.

But the point of origin of the tabetic process is still in dispute. The old theory that the fibre degeneration is secondary to ganglion cell changes has been refuted by the works of Marinesco (35), Thomas and Hauser (22).

Vulpian, quoted by Homén (2), considers the intramedullary lesion a Wallerian degeneration, caused by a neuritis of the posterior roots.

Marie and Guillain (18), after taking into consideration certain facts concerning the posterior lymphatic system—to which we have referred above—state that tabes is a lesion of this system, most probably of syphilitic origin.

In 1897 Philippe (3), in his article on the subject of tabes, said that to describe it as a sclerosis of the posterior columns and roots was insufficient; in fact he considered the interstitial lesions to be entirely secondary. His examination of the cords of early and advanced tabetics led him to the conclusion that the lesion of tabes begins as a primary parenchymatous degeneration, which attacks the medium length fibres first, and that this degeneration commences in the roots or at the point where the fibres enter the cord. The roots, in his cases, were certainly diminished in size, but he adds that this diminution was due to an atrophy of the fibres, that the interstitial tissue was normal, and that the interannular nuclei were not increased in number.

The atrophy of the roots shows, however, that he was not dealing with an early stage of the lesion.

Gowers (23) also has found that in cases in which the cord was much affected, the roots showed wasting of the nerve fibres and only slight increase of connective tissue.

The importance of a lesion of the posterior roots as the starting point of the degeneration in tabes has been much developed by Nageotte (24). He attributes the degeneration of the fibres in the posterior columns to a transverse interstitial radicular neuritis, which is an exaggeration of a diffuse meningitis, and which attacks the posterior roots close to the ganglion. This begins as a perineuritis, spreads inwards, and gives rise to an endoneuritis. At the seat of inflammation the injury to the fibre shows itself as a local atrophy, and although this is not sufficiently severe to kill the fibre, yet its presence produces a lowering of vitality, and induces a degeneration, which, he asserts, begins at the peripheral end of the neuron and spreads backwards towards the cell of origin. In course of time this degeneration reaches the seat of the neuritis, and then its starting point can no longer be distinguished.

Nageotte's theory cannot hold for all cases of tabes as

this local neuritis is not a constant factor, nor even in advanced stages of the disease does one constantly find changes in the extramedullary part of the root at all comparable—in kind and degree—to those situated within the cord. In fact, there is sufficient evidence to show that often in the early stages of the disease no appreciable changes can be demonstrated in the roots, and that when they do appear in the later stages, they not only are greatly exceeded by the intramedullary lesions, but they also differ in character from them.

Thomas and Hauser (22), while ascribing the fundamental lesion of tabes to a neuritis of the posterior roots above the ganglion, admit that the lesion is more a distrophy than an inflammation; nor can the atrophic non-inflammatory condition of the roots in Philippe's cases be overlooked (*loc. cit.*).

Williamson (25) also, in 1897, published the record of a case of tabes, which had lasted for three years, in which the sclerosis in the posterior columns of the cord was well marked, while in the posterior roots a negative result was obtained by the Marchi and Weigert methods, and there was a doubtful increase in the quantity of interstitial tissue present. In any case, there were no lesions in the roots which could have produced the marked sclerosis in the posterior columns.

We have in cases of marked sclerosis of the root-entry zones failed to find sclerosis in the roots sufficient to account for the intramedullary lesion. This, together with the fact that numerous authors have observed extensive disease of the exogenous system of the posterior columns independent of root degeneration in conditions other than tabes, shows that changes in the extramedullary part of the sensory protoneurons are not essential for the causation of the cord lesions, and when present are secondary.

All these theories, excepting that of Nageotte's, have been based upon the study of tissues taken from tabetics in whom the disease had reached a considerable degree of chronicity, and we hold with Philippe (3), that under such circumstances it is impossible to determine exactly where the degeneration of the sensory protoneurons has commenced,

owing to the secondary changes induced by the degeneration of the nerve fibres. It is the absence of these secondary changes which make the study of acute system lesions in the posterior columns so profitable, because we are able to locate accurately the starting point of the lesion and to draw important deductions.

It seems to us that there is no valid reason why the tabetic process should not begin at the same point. In fact, there is a certain amount of direct, as well as much indirect, evidence that it does so, because in early cases the changes found in the posterior roots are insufficient to account for the intramedullary lesion.

Homén (2) has summarised the subject of the pathogenesis of tabes in so thorough a manner that his remarks are well worth giving at some length. In early cases he has found that the process begins in the intramedullary part of the fibres, and the first changes are parenchymatous. He admits the difficulty of determining precisely where the process begins, but sometimes, quite at the commencement of the disease, the changes are confined to the intramedullary part of the root, and often to the point of entry into the cord or close to it. He suggests that the posterior columns are specially vulnerable because in cases of increased cerebral pressure, and in various cachexias, the intramedullary portion of the root fibres is alone affected. To account for this vulnerability he suggests that these fibres are more open to attack by their numerous bendings in the cord, by their passage through the pia where they may be subjected to mechanical injury (Obersteiner and Redlich), and because toxins pass upwards more readily by the posterior roots. At the same time he fully recognises the importance of the loss of the neurilemma.

The degenerative process having once been established, the further spread in the already weakened neuron takes place, (1) by the continued action of the original poison on the nerve, carrying on the degeneration; (2) by the production of a secondary degeneration in the distal part of the neuron (either by a direct spreading along the fibre or as a consequence of disturbance of the trophic interdependence in

the different parts of one and the same neuron); (3) by the simultaneous action of both these factors.

The further development consists in the involvement of new neurons in the same root and gradually of other roots.

Gradually the neuroglia, vessels, and lymph channels are affected by the substances set free by the destruction of nerve fibres, and in the late phases the lesion spreads into contiguous areas with the result that the systemic character of the lesion may be somewhat altered. Homén refutes the compression theory of Obersteiner and Redlich on the ground that in early cases meningeal changes are absent.

Homén's view as to the point at which the degeneration starts has been long held by Gowers (23), who found that the sclerosis was most dense in the part adjacent to the posterior cornu, through which the posterior fibres pass, and near the surface of the cord.

If we accept that tabetic lesions are primarily parenchymatous and begin at the point to which we and others have drawn attention, and consider in this connection the results obtained by Köster (26) after division of the posterior roots, then the reason why these should show no change in early cases of tabes will be evident, as also the reason why—as the disease progresses—this part of the sensory protoneuron should only undergo alteration slowly, and never exhibit changes equal in degree to those of the intramedullary part.

The accepted view that division of the posterior root produces no alteration of the spinal ganglion cells must be rejected after Köster's experiments. He has shown that division of the posterior roots causes a reaction in the spinal ganglion cells—much later than after division of the peripheral process—which induces in some cells a simple atrophy. and in others complete degeneration.

As a consequence of death of some cells the Marchi method gives a positive reaction between the point of division and the ganglion, and with simple atrophy of other cells—without any structural alteration—atrophy of the fibres must follow.

Degeneration of the axis-cylinder at the cord margin

—Erbslöh (11)—is the equivalent of Köster's experiment, with this difference, however, that in tabes all the fibres are not attacked and do not degenerate at the same period. A few are first affected, and the disease more or less gradually spreads to others. The fibre degeneration and atrophy in the posterior roots as a result of reaction in the spinal ganglion cells must, therefore, develop much more slowly than after experimental division, and, from what we know of the results of division of the peripheral process of the cell, the minority of cells will undergo complete degeneration, while the majority, recovering from the reactive phase, will remain permanently atrophied. Such seems to us a reasonable explanation of the comparative integrity of the posterior roots, even when the entry zones in the cord are sclerosed.

So far, in this paper, we have only dealt with what appears to us to be the exciting cause of posterior column lesions, and also with the anatomical structure of the fibres at the vulnerable point, which, we consider, is of great importance in their production.

We have also mentioned that in the cords which we have examined, the fibres in the cervical and lumbo-sacral enlargements were affected more than these in the dorsal region; and, moreover, in a given root, only some of the fibres had been attacked.

This has been seen in various cases of general intoxication, and it holds good also in tabes, and it is this fact which determines the varieties of syndromes, which are met with in the early stages of this disease.

This selection depends, in all probability, on some developmental anomalies, or other predisposing causes, which have rendered some fibres more liable to degenerate under the action of a poison, rather than to any "selectivity" on the part of the poison, as has been so often suggested.

With regard to the predisposing causes, there can be no doubt that the most important is syphilis. But, although it must be admitted that syphilis plays a very important rôle in connection with lesions of the central nervous system, the opinion that it is the direct exciting cause is not so strongly held as it was a few years ago, and we find that

Homén, Cassirer (27), and many others now speak of it as "the most important predisposing cause." In this connection Gowers (23), while recognising the importance of syphilis as a factor in the determination of the disease, has suggested that there is a special liability to degeneration in the elements which suffer.

Moreover, many cases have been published in which syphilis could be excluded, and the lesions of the posterior columns has been attributed to trauma, cold [von Leyden (28), Windscheid (29)], malaria, alcohol, influenza [Maurice Faure (30)], saturnism, pellagra, ergotism [Pansini (31)].

Of considerable importance also are the developmental anomalies, which are commonly seen in the spinal cords of those who suffer from spinal diseases. These anomalies, which are produced in intra-uterine life, may have been insufficient to give rise to any symptoms; but, according to many authorities, they indicate a diminished resistive power of the fibres.

Rebizzi (32) has described a case in which he found a heterotopia and a neuroma in the posterior columns of a cord, and there were present signs of an incipient tabes also. We, too, have seen developmental anomalies in the posterior columns and amongst the cells of the posterior root ganglia in general paralytics and imbeciles.

But it is also possible that the injury to the nerve fibres has been so slight that it cannot be detected by the ordinary means of investigation, but yet it may have been sufficient to produce a lowered power of resistance. Thus, we find Sciuti (33) stating that "the lesion in tabes consists of changes in fibres which started with some defect"; and Homén (34), in his article on Friedreich's disease, said, "we have to do with a weakened or anomalous condition of certain neuron systems, whose resistance against usual demands is lowered." Similar weakened conditions may be produced by the acute infective diseases, which are so common in early life, on the one hand, and by prolonged strain on the other hand.

Such, then, are the developmental anomalies and predisposing causes which assist in the determination and

localisation of the lesions of the spinal cord, and which must be taken into consideration with the other causes and conditions, already referred to in this paper, when we attempt to explain the pathogenesis of these lesions.

But granted a hereditary or acquired weakness of the sensory protoneurons and a determining exciting cause, there still remains to be explained the fact, that intramedullary system lesions of these fibres begin constantly at the vulnerable point.

We consider that it is the loss of the neurilemma which renders the intramedullary portion of the sensory protoneuron more open to the attack of noxious agents. The neurilemma affords to the extramedullary part a protective influence which, in all probability, is more vital than mechanical; a view which is supported by the observations of many who have studied nerve regeneration.

Tabes, then, is a system lesion, which begins as a parenchymatous degeneration of the sensory protoneurons starting at the point where the neurilemma is lost. We thus agree with Gowers (23) when he says that "in system diseases the primary change is, as a rule, in the nerve elements and the overgrowth of interstitial tissue is secondary."

References.

(1) Orr and Rows. *Brit. Med. Jour.*, October 15, 1904.
(2) Homén. "Tabes Dorsalis." *Handbuch der patologischen Anatomie des Nervensystems* (Flatau, Jacobsohn, Minor, 1904).
(3) Philippe. *Archiv. de Neurol.*, No. 21, 1897.
(4) Edinger. Vorlesungen über den Bau der nervösen Zentralorgane des Menschen und der Thiere, 1904.
(5) Obersteiner. "The Anatomy of the Central Nervous System in Health and Disease." 1900, p. 228.
(6) Nageotte. *Rev. Neurol.*, No. 1, 1904.
(7) Lugaro. "Allgemeine patolog. Anat. der Nervenfasern." *Handbuch der patologischen Anatomie des Nervensystems* (Flatau, Jacobsohn, Minor, 1904).
(8) Bruce. *Rev. Neurol.*, No. 14, 1900.
(9) Homén. *Rev. Neurol.*, No. 14, 1900.
(10) Williamson. *Brit. Med. Jour.*, January 16, 1904.
(11) Erbslöh. *Monats. für Psych. u. Neurol.*, Bd. xiii., H. 2, 1903.
(12) Homén. *Rev. Neurol.*, No. 20, 1900.
(13) Erb, Tizzoni, Neumann. *Patologia Generale*, Lustig, 1901.

(14) Mönckeberg and Bethe. *Archiv. f. mikr. Anat.*, Bd. 34, H. 2, 1899.
(15) Ballance and Stewart. "The Healing of Nerves," 1901.
(16) D'Abundo. *Annali di Nevrologia*, f. 3-4, 1896.
(17) Guillain. *Rev. Neurol.*, No. 23, 1899.
(18) Marie et Guillain. *Rev. Neurol.*, No. 2, 1903.
(19) Homén. *Arbeiten aus dem patolog. Institute zu Helsingfors*, 1902.
(20) Orr. *Review of Neurology and Psychiatry*, October, 1903.
(21) Spiller and Frazer. Reviewed in *Rev. Neurol.*, No. 12, 1904.
(22) Thomas et Hauser. *Nouv. Icon. de la Salpêt.*, 1902.
(23) Gowers. "Diseases of the Nervous System," 1892.
(24) Nageotte. *Soc. de Biologie*, April 7, 1900, C.R., p. 354; *Presse Médicale*, No. 99, 1902; *Presse Médicale*, No. 1, 1903; *Nouv. Icon. de la Salpêt.*, January-February, 1904.
(25) Williamson. *Med. Chron.*, October, 1897.
(26) Köster. *Neurol. Centralblatt*, No. 23, 1903.
(27) Cassirer. "Tabes und Psychose," 1903.
(28) von Leyden. *Berliner klin. Wochen.*, No. 20, 1903.
(29) Windscheid. *Münch. medizin. Wochen.*, Nos. 26-30, 1903.
(30) Maurice Faure. *Rev. Neurol.*, No. 16, 1902.
(31) Pansini. *Riv. critica di Clin. Med.*, Nos. 1-2, 1902.
(32) Rebizzi. *Riv. de patol. nerv. e. ment.*, F. 10, 1903.
(33) Sciuti. *Annali di nevrolog.*, fasc. 6, 1901.
(34) Homén. "Friedreich'sche Krankheit oder Ataxie." *Handbuch der patol. Anat. des Nervensystems* (Flatau, Jacobsohn, Minor, 1904).
(35) Marinescò. *Rev. Neurol*, No. 23, 1900. *Presse Médicale*, No. 62, 1901.

THE FALSE LOCALISING SIGNS OF INTRACRANIAL TUMOUR.

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THIS communication is based upon the results of 161 consecutive cases of intracranial tumour which have been examined in the Pathological Laboratory of the National Hospital during the years 1894-1904. The examinations were made by Dr. Colman, Dr. Risien Russell, Dr. Batten, Dr. Farquhar Buzzard, and by the writer of this paper.

The majority of these cases came under the writer's personal clinical observation. His best thanks are here expressed to his colleagues upon the staff of the National Hospital, to Dr. Colman, and to Dr. Farquhar Buzzard, Pathologist to the Hospital, for their kind permission to make use of the clinical and pathological notes upon these cases.

The object of this paper is to show :—

(1) That local signs appearing late in the course of intracranial tumour, where general signs alone have pre-existed, are often of false portent.

(2) The relative frequency with which local signs have been due in this series of cases to the presence of vascular lesions, meningitis, hydrocephalus, local spreading œdema of the brain, secondary deposits of new growth, and posterior degeneration.

(3) That the absence of usually accepted local signs during the early days of illness in intracranial tumour is in itself a most important localising indication, confining the disease to the supratentorial region.

(4) That true localising signs at one time present may later become concealed or undemonstrable owing to the

development of other signs, and that in cases which come under observation for the first time late in the disease, diagnosis may be difficult, erroneous, or impossible.

The relative frequency with which the different parts of the brain were the seat of new growth in this series of cases was as follows:—

Frontal	24
Central	20
Parietal	7
Occipital	3
Temporo-sphenoidal	12
Centrum ovale	17
Basal ganglia	14
Brain stem and pons	22
Cerebellum	26
Other situations	16
Total	161

The False Portent of Late Localising Signs.

The meaning which the writer wishes to convey by this heading will be at once apparent in the following examples:—

A man, aged 20 years, suffered with headache, vomiting, and optic neuritis. Seven months after these symptoms appeared he was totally blind from post-neuritic atrophy. In the fifteenth month of his illness localising signs began to appear. In the seventeenth month his condition was as follows:—Paralysis of left external rectus. Marked nystagmus with the slow movement to left. Complete nerve deafness of left ear. Left peripheral facial paralysis. Left cerebellar position of head (head inclined to left shoulder, and face rotated to right). Marked head retraction during paroxysms of pain. Bilateral ataxy left > right, with lurching to left on the attempt to walk. Knee-jerks: left, normal; right, diminished. Double extensor response. His condition remained unchanged until death in the twentieth month.

The localising symptoms were apparently conclusive of a growth in the left posterior fossa involving the cerebellum and the sixth, seventh and eighth nerves. The autopsy

revealed a glioma of the left prefrontal region. The cranial nerves seemed normal to the naked eye, but the intramedullary portion of the left eighth nerve showed degeneration. The cerebellum was much indented by the edge of the foramen magnum, the left lateral lobe forming more of the pressure cone[1] than the right.

In this case the entire absence of localising symptoms during the first fifteen months of illness, when several general symptoms were present, not compatible with the diagnosis of tumour in any but a "silent" region of the brain. The late development of signs indicative of a lesion in the posterior fossa was the indirect and secondary result of a tumour situated at a distance.

A child, aged 8 years, suffered with headache and vomiting, and developed, within a few weeks, well-marked optic neuritis, and signs of a left-sided cerebellar lesion. These signs persisted with exacerbations and remissions for twelve months, when convulsive seizures, commencing in the left hand and being confined to the left arm, occurred, without loss of consciousness. The Jacksonian attacks were repeated at intervals, and in some of the later attacks the convulsion became general and consciousness was lost. The child died after an illness of eighteen months. A subtentorial tumour was found, which had probably arisen in the left cerebellar hemisphere, and had extended into the right cerebellar hemisphere. The fourth ventricle was much occupied by growth, and the aqueduct was obstructed. The ventricles were considerably distended, and the dilatation was more marked in the central region of the right lateral ventricle. There was no local lesion in the right central region that would account for the Jacksonian attacks other than the thinning of the ventricular wall.

This case shows that local convulsions are not always of localising value, and that they may be symptomatic of ventricular distension.

[1] In many cases of intracranial tumour of long duration, it is found *post mortem*, that the posterior and inferior parts of the cerebellum have become pushed down and backwards into the foramen magnum, and the medulla itself being somewhat caudally displaced, the two structures together forming a cone-shaped plug tightly filling up the foramen magnum.

False localising signs were met with in twenty cases out of 161 cases in this series = 12·5 per cent. In two cases the false localising signs were due to vascular lesions (hæmorrhage, one case and thrombosis one case), situated at a distance from the growth.

In the rest of the cases the false signs were attributable to the indirect intracranial results of intracranial new growth. They were met with in 13 per cent. of the supratentorial tumours, and only twice among fifty-four cases of subtentorial tumours.

The following localising signs occurred either singly or in combination as false signs :—

Paralyses of cranial nerves.
Hemianopia.
Jacksonian epilepsy.
Bilateral spastic paresis.
Cerebellar signs.

Paralysis of the Cranial Nerves.

The Olfactory Nerves.—Anosmia was only found in this series, as the direct result of local pressure from the growth, upon the olfactory tract and bulb. There was not the least evidence of the occurrence of olfactory neuritis comparable with optic neuritis, as some authors have argued. The writer has, moreover, examined a large number of cases of long-standing intracranial tumour, in which optic neuritis has caused blindness, and has not found anosmia to be more frequent among them than among patients suffering with diseases other than of the nervous system.

Hemianopia was met with in two cases.

In one case the tumour was situated in the left upper central region and was of large size. The absence of the hemianopic pupillary reaction suggested that the right hemianopia present was due to interference with the left optic tract. The hemianopia made its appearance, together with paralysis of the left fifth and right sixth nerve, four and a-half months after paralysis of the right leg had become complete. There was no *post-mortem* evidence of abnor-

mality of any part of the optic path or of the fifth and sixth nerves.

In the second case right tract hemianopia and left trigeminal paralysis and anæsthesia appeared four and a-half years after the onset of the earliest symptoms in a case of left prefrontal tumour.

The association of right tract hemianopia with left trigeminal palsy strongly suggested that preponderant indirect pressure upon the left middle fossa by a tumour in the left cerebrum had determined the combination.

Paralysis of the third nerve was met with in two cases. It was associated with paralysis of the sixth nerve, and appeared later than the sixth nerve paralysis.

One of the cases was that of a large growth in the subcortex of the right temporo-sphenoidal lobe. Seven months after the development of optic neuritis, paralysis of the right sixth nerve appeared, followed in a few days by paralysis of the left sixth nerve. Three weeks later paralysis of the right third nerve appeared, followed in a few days by paralysis of the left third nerve.

The second case was that of a large prefrontal growth upon the left side. Nine months after the onset of symptoms the left sixth nerve became paralysed, then the right sixth, and, some time later, the left third nerve became paralysed.

The important facts which these two cases illustrate are: (1) That the third nerve palsy occurred subsequently to paralysis of the sixth nerve, and (2) that the nerves were affected earlier upon the side of the lesion.

If the paralysis of these nerves is the result of shifting backwards of the brain stem from supratentorial pressure, the effect being traction upon the nerves attached to the brain stem, in proportion as their direction is more nearly a frontal-caudal direction, then the shifting backwards of the brain stem would cause paralysis of the sixth nerve first, then of the third nerves, and lastly of the seventh and eighth, while the nerves which are transversely directed or with sinuous course would be little affected. Such traction would obviously be first manifested upon the side

where supratentorial pressure is greatest, that is upon the side of the lesion.

Paralysis of the fourth nerve was not met with as an indirect effect of intracranial tumour. The course of this nerve from its origin to its point of passage through the dura mater is so sinuous that traction could hardly be exerted upon this nerve by backward shifting of the brain stem.

If it be argued that fourth nerve paralysis is difficult to determine and may have been overlooked, it can be stated with certainty that fourth nerve paralysis never occurred as an isolated occular palsy in this series of cases. Every case in which diplopia occurred was carefully investigated, and the diplopia of superior oblique palsy is so distressing as to render impossible its being overlooked in a patient who is fully conscious and who is not blind.

Paralysis of the sixth nerve was met with in twelve cases of supratentorial tumour. It was unilateral in six cases, and bilateral in six cases. The paralysis when unilateral was right-sided in four cases and in two cases left-sided. It is therefore, more common as an indirect result of intracranial tumour than paralysis of any other cranial nerve. This fact has been recorded by the majority of observers, and the explanation has been given that the sixth pair of cranial nerves are more liable to be affected as a result of general increase of intracranial pressure on account of their long intracranial course and exposed position. I am unable to accept this explanation since the intradural position of the nerve alone is exposed to the intracranial pressure, the portion between this and the sphenoidal fissure being protected by the dura mater and by the blood of the cavernous sinus.

In many cases of supratentorial tumours the tentorium is pressed downwards, and the brain stem and cerebellum are also pressed downwards. The medulla and posterior part of the cerebellum come to lie partly within the foramen magnum, the cerebellum being deeply indented by the edge of the foramen magnum so that these structures together form a conical plug which fills up the foramen magnum. This alteration in the position of the brain stem may be also

demonstrated *post mortem* by the deep indentation of the crura cerebri by the free edge of the tentorium.

All the cranial nerves attached to the brain stem, with the exception of the sixth pair, are directed transversely or obliquely from their superficial origins to their exits from the subdural space. The sixth pair of nerves, however, are directly straight forwards. The shifting backwards of the brain stem can have little traction effects upon the roots of those nerves, which are directed transversely, but considerable traction effect must occur from such shifting in the case of the sixth nerves.

I wish to submit it is from this cause that sixth nerve paralysis comes to be the most frequent cranial nerve palsy, resulting indirectly from the presence of intracranial tumour.

The fifth nerve was affected in two cases, and in both of these tract-hemianopia was also present. The tumours were large and were situated in the left prefrontal region and left frontal centrum respectively. In both cases the motor paralysis appeared before the sensory paralysis.

The fifth nerve paralysis and the hemianopia probably resulted from the indirect pressure of the growth upon the middle fossa of the same side.

The sensory loss appeared in the area of distribution of the three divisions of the trigeminus in the order 3, 2, 1.

The Seventh Nerve.—Peripheral facial palsy occurred in two cases. It was preceded by paralysis of the sixth nerves. In one case a large tumour occupied the posterior horn of the left lateral ventricle; the facial paralysis was upon the left side and was not associated with paralysis of the left eighth nerve. In the second case a large tumour was situated in the left frontal region, and complete paralysis of the left seventh and eighth nerves occurred.

It may be here remarked that slight weakness of the lower face is of very common occurrence in supratentorial tumours of any situation. It is not associated with any alteration of electrical excitability. The writer has no explanation for its occurrence to put forward.

The Eighth Nerve.—Bone deafness was present in two cases. It occurred once in connection with paralysis of the

seventh nerve in the above-mentioned case of frontal tumour and once without facial paralysis in a case of temporo-sphenoidal growth.

The writer's experience directly negatives the theory that auditory neuritis in any way comparable with optic neuritis occurs in intracranial tumours. Excluding these two cases, and those cases where the auditory path was directly involved by growth, he has found that defects of hearing are not more common in long standing cases of intracranial tumour than in patients suffering with diseases other than of the nervous system.

The ninth, tenth, eleventh, and twelfth nerves were not indirectly affected in any of the cases.

Speaking collectively therefore, palsy of the cranial nerves, as an indirect result of intracranial tumour, is not of very common occurrence. It was present in sixteen cases out of 161 cases, or 10 per cent. The possible fallacy of statistics was strikingly shown in this series. Of the cases occurring between 1894 and 1899 it occurred in fourteen cases out of ninety-two, equal to 15 per cent., while of the cases between 1900-1904, it occurred in two cases out of sixty-nine, under 3 per cent. One possible reason for its rarity of late years is that cases of intracranial tumour are operated upon earlier and more frequently than was formerly the case. In all the cases the cranial nerve paralysis has been a relatively late symptom.

The determination as to what period is "early" or "late" depends upon the severity of the symptoms and the rapidity of growth of the tumour. A local sign appearing three months after the onset of symptoms would be a late sign in a case where the general signs were of great and persistently increasing severity; and on the other hand, it would be an early sign in a case where the general signs were not obtrusive and increased slowly. Paralysis of the external rectus was observed as early as the sixth week in a case with severe general signs. This patient died on the tenth week, and a tumour the size of a large orange was found, the pressure signs being very well marked.

The cranial nerve palsies once appearing are generally permanent, but this is not always so. Paralysis of the sixth nerve has disappeared in two cases, and in one case paralysis of the fifth nerve, at one time complete, recovered to a great extent. In such cases it is possible either that there is some alteration in the mechanical interference with the nerves in the course of time, or that they become accustomed as it were to the changed conditions.

THE OCCURRENCE OF JACKSONIAN EPILEPSY, HEMI-EPILEPSY AND GENERAL CONVULSION IN CONNECTION WITH TUMOURS OF THE BRAIN STEM AND CEREBELLUM.

In two cases of tumours of the cerebellum local convulsion of slow spread, and confined to the arm and face, were repeatedly observed. Both were cases of long standing, and in both slight signs of bilateral spasticity were present. In the early stages of the illness the signs of cerebellar involvement were conclusive. The autopsies showed that considerable ventricular distension was present, but there was no other lesion present to account for the local convulsion.

Hemi-epilepsy was observed in one case of glioma of the pons and in one case of cerebellar tumour, while general convulsion occurred in several cases of tumour of the brain stem and of the cerebellum.

In all these cases the symptoms were of long standing when the convulsions first occurred, and in all of them the autopsies revealed considerable ventricular distension.

These remarks have reference to local convulsion of the cortical type and these must not be confused with certain local convulsions of a peculiar nature and order, which undoubtedly occur in lesions of the pons and cerebellum, and which have their origin in these structures. Such "lowest level fits" have been long since described by Dr. Hughlings Jackson. Most interesting and important observations upon local convulsion of cerebellar origin are published in this number of BRAIN by Dr. Grainger Stewart and Dr. Gordon Holmes, who have given me the privilege of acquaintance with their observations while in progress.

That hydrocephalus alone is capable of causing local convulsion is proved by the not rare occurrence of the phenomenon in cases of primary hydrocephalus.

Further, if the weighty arguments of Dr. Hughlings Jackson be followed, that every epileptic seizure has of necessity a local commencement, the distinction between local and general convulsions as local signs of organic disease of the brain becomes invalid.

It is, I think, a very commonly accepted fallacy that local convulsion is a sure sign of local gross lesion of the cerebral cortex. The commonest cause of Jacksonian convulsion is probably idiopathic epilepsy. Of the patients who have attended the out-patient department at Queen Square during the past five years, and who have suffered with local convulsion, the majority have been cases of idiopathic epilepsy, provedly so by the history and course of the malady; while a minority have been cases of local gross disease of the cerebrum.

On the other hand, patients who have attended with symptoms apparently conclusive of idiopathic epilepsy and who have later on developed symptoms of intracranial growth have been numerous.

It is submitted, therefore, that the occurrence of local convulsion of hemi-epilepsy and of general convulsion, when presenting for the first time, long after the general signs of intracranial growth have appeared, is to be disregarded as a localising sign. It is the result of secondary hydrocephalus, and the latter may result no matter where the growth may be situated within the skull. Slight bilateral spasticity is not infrequently an indication of the existence of such ventricular distension.

Bilateral spastic paresis occurred late in several cases where there was not involvement of the pyramidal tracts neither directly nor by pressure. It amounted to clumsiness of movement in the limbs, slight rigidity, increase of the deep reflexes, foot clonus, and the extensor response in the plantar reflex. In all the cases there was considerable distension of the lateral ventricles. Bilateral spastic paresis thus occurring in the late stages of intracranial tumour is

to be ascribed to the cortical wasting which results from secondary distension of the ventricles and perhaps also to degeneration of the cortical elements, which the chronic partial evascularisation consequent upon slowly increasing intracranial pressure must entail.

The occurrence of bilateral spastic paresis in cases of intracranial tumour usually indicates that the growth is situated in some region where the pyramidal tracts are close together; in other words, that the growth implicates the brain stem. It is especially in connection with infiltrating gliomata of the brain stem that this sign is met with.

The series of cases here dealt with show that bilateral spastic paresis may occur as a late sign in cases of intracranial tumour wherever the growth is situated, provided that distension of the ventricles occurs. Now secondary hydrocephalus may occur with a long standing growth in any situation.

It has been already pointed out that palsy of certain cranial nerves is of frequent occurrence as an indirect result of long standing intracranial growth.

The combination of bilateral spastic paresis with cranial nerve palsies, usually a sure sign of implication of the brain stem may therefore be produced in the late stages of a growth in any situation within the skull. The determination as to whether this combination is a true or a false localising sign is sometimes difficult. The reasons for the difficulty, and the points which may serve in the discrimination, will be apparent from the following table :—

	Glioma of Brain Stem.	Late Pressure Effect.
Bilateral spastic paresis	Occurs early or late ...	Occurs late.
Degree	Slight or severe, often unilateral for a time	Bilateral.
Cranial nerve palsy ...	Almost always of nuclear type at first	Always of peripheral type
	Any nerves may be involved	Never the 4th, 9th, 10th, 11th and 12th.
	Optic neuritis often absent	Optic neuritis always severe.

The question as to whether signs significant of cerebellar lesion result from the distortion of the posterior parts of the lateral lobe of the cerebellum, in the formation of the pressure cone, in cases of long standing intracranial tumour, is one of great difficulty.

The writer's opinion is that such signs are of not infrequent occurrence. Such signs must of necessity be bilateral since both lateral lobes are distorted. Further, the signs of cerebellar disease are for the most dynamic signs, and for their elicitation it is necessary that the patient shall be in possession of his mental and physical powers. For an instance, it is impossible to detect the presence of bilateral ataxy with certainty if bilateral paresis be present, or if the patient is mentally unable to attend and obey commands. Similarly the gait and tendency to fall in a particular direction cannot be tested in a patient who cannot stand and walk, or who is blind. In the late stages, therefore, of long standing intracranial tumour the tests for cerebellar involvement are often inapplicable.

In many cases, however, of long standing intracranial growth, nystagmus is met with, unilateral or bilateral ataxy may occur, the patient's gait and attitude may suggest cerebellar disease and he may constantly fall in a particular direction and in such cases no other abnormality of the cerebellum has been found.

The writer has had the opportunity of examining a very large number of cases of cerebellar tumour and is of opinion that tumours of the cerebellum rarely fail to show the signs of cerebellar involvement, as described by Risien Russell, early in the course of the case. The same holds good for all cases of subtentorial growth that they give early localising signs, with the distinct exception of some cases of glioma confined to the pons.

If this opinion be correct, the appearance of signs indicative of cerebellar involvement, late in the course of a case where no signs of subtentorial growth had previously existed, should not be allowed much weight in local diagnosis.

The absence of the usually accepted localising signs for many weeks or months after the general symptoms have

become severe is in itself of very important localising significance.

Since the supratentorial division of the intracranial cavity is roughly seven times as large as the infratentorial division, other things being equal, a non-localisable tumour has a probable supratentorial situation in seven cases out of eight. In this series the proportion of supratentorial tumours to infratentorial tumours has been rather more than three to one.

While "silent areas" form a large proportion of the cerebral hemispheres, and the possibilities of compensation are considerable in this region, below the tentorium the tracts connecting the brain with the periphery are densely grouped together and further, it appears that lesions of the cerebellum are almost always early productive of characteristic symptoms. During the last five years, of a very large number of cases of cerebellar tumour in the wards of the National Hospital, there was no case verified by operation or by necropsy that had not been correctly diagnosed during life.

It appears, therefore, that a tumour which, while giving rise to marked general symptoms, affords no localising symptoms for many weeks, may be located alone the tentorium.

The Production of Symptoms from Other Lesions Co-existing with a Neoplasm.

Meningitis.

Sir William Gowers draws attention to the frequency with which some degree of meningitis co-exists with intracranial tumour, more frequently in the immediate neighbourhood of a growth which implicates the meninges, but sometimes also existing in isolated patches at a distance from the growth. With the exception of one case of tuberculous tumour of the cerebellum, in which tuberculous meningitis supervened, inflammation of the meninges was not found in this series of cases.

Vascular Lesions.

The occurrence of vascular lesions in regions remote from the situation of the tumour may give rise to prominent localising signs which are likely to lead to a false diagnosis, as in the following case: A man, aged 39 years, was admitted into the National Hospital on July 16, 1900, under the care of Dr. Hughlings Jackson. He had suffered with headache and vomiting for two months, and he presented well marked optic neuritis. Two days before admission he had a fit, commencing upon the right side, in which he lost consciousness. On recovering consciousness he presented aphasia and weakness of the right arm and face. After admission the paralysis of the right arm and face became progressively worse. An exploratory operation in the left fronto-central region revealed an extensive softening. Subsequently the necropsy showed that a large tumour was situated on the right optic thalamus, and that the anterior branches of the left Sylvian artery were extensively thrombosed.

While it seems impossible to avoid diagnostic error in cases of this nature, yet it is advisable to bear in mind the possibility of vascular lesions in cases of intracranial tumour above middle age, and who have hard arteries.

In one case of frontal tumour a hæmorrhage the size of a walnut was found *post mortem* on the right lateral lobe of the cerebellum. It was a very recent hæmorrhage and had probably occurred just before death, for no corresponding symptoms were observed. A similar hæmorrhage into the cerebellum was found in a case of rapidly fatal hydrocephalus in an adult; again the hæmorrhage was very recent and no symptoms were referable to its occurrence.

It has been stated by many authorities that the presence of a tumour upon one of the larger cerebral arteries may cause extensive thrombosis in the region of its distribution. In this series tumours have often existed in close proximity with the larger arteries but thrombosis has not been met with. It may be pointed out that the pressure which a tumour can assert is governed by, and cannot exceed, the maximum of the cerebral arterial pressure.

Local Spreading Œdema.

The occurrence of cerebral œdema in the immediate neighbourhood of cerebral neoplasms is well known. It is referred to here as the probable cause of acute symptoms in two cases among this series; the patients, while in apparent good health, were seized with urgent and rapidly fatal symptoms.

A girl, aged 23 years, came as an out-patient to the National Hospital in May, 1897. She had been perfectly healthy till May, 1895, when she had three sudden attacks of loss of consciousness at intervals of a few days and in the third of these attacks she was slightly convulsed. For the following year she suffered with occasional attacks of giddiness associated with pallor, and it was for such attacks that she sought advice. She presented no sign of organic disease. She was treated with bromides and her attacks of giddiness became less frequent. She came regularly to the out-patient department for the next eighteen months, during which time her health was good and she continued her work. On November 12, 1898, she was apparently in excellent health. The following morning she was found to be very somnolent. The somnolence deepened and she was admitted as an in-patient into the National Hospital, under the care of Dr. Bastian, on November 14. When admitted she was evidently very ill physically and she lay in bed in a half conscious state and never moved a muscle unless roused. Early optic neuritis was the only other abnormal physical sign that could be detected. Two days later paralysis of both sixth nerves made its appearance. The optic neuritis became severe, the coma deepened, and she died on the fourteenth day of her illness. The autopsy was performed by Dr. Risien Russell. A small, hard, thickly encapsuled tumour, evidently of long standing, was found, involving the second and third left frontal convolutions and the posterior part of the orbital lobule. The measurements of the growth were $5 \times 3 \times 3$ cms. For a considerable distance round the growth the white matter of the centrum semi-ovale was much swollen. It was much

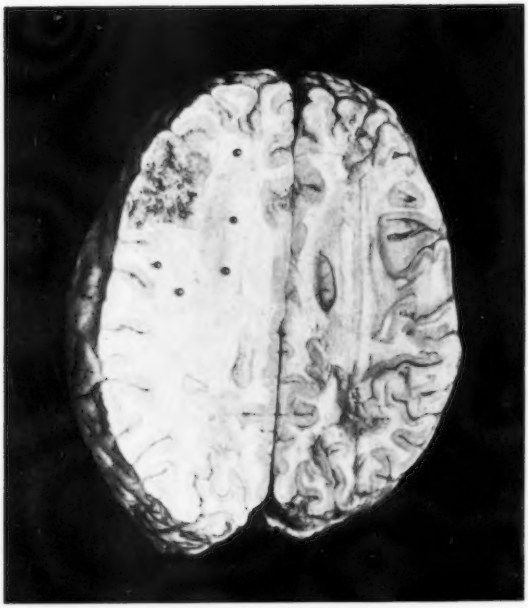

FIG. 1.

Acute œdema in the region surrounding a hard, slowly-growing tumour.

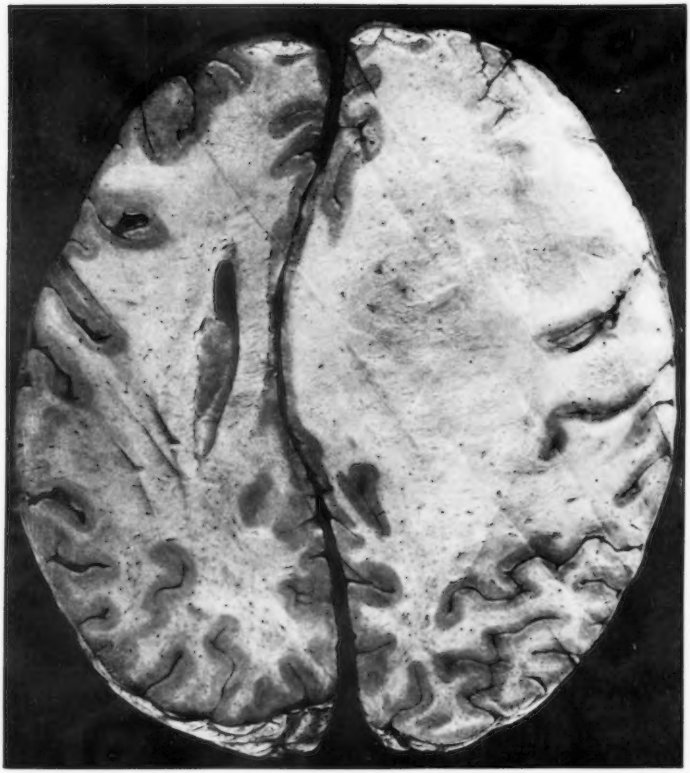

FIG. 2.

Œdema of the centrum semiovale, from a small nodule in the right prefrontal cortex, secondary to a renal carcinoma.

softer than normal and was almost translucent. Examined microscopically this swollen white matter presented marked vacuolation and lymphatic distension as the only peculiarities (fig. 1).

In this case one cannot but conclude that the epileptic symptoms were referable to the presence of the growth, and that the urgent and rapidly fatal symptoms were the result of the spreading œdema.

The second case in which symptoms were referable to spreading œdema, was one in which, as a result of primary renal carcinoma, a secondary tumour about the size of a walnut occurred in the cortex of the right prefrontal lobe. The cerebral symptoms were very urgent and death occurred in fourteen days after their appearance. There were no localising signs during the first seven days. Afterwards, some degree of hemiplegia appeared.

The right hemisphere was found to be greatly increased in size and its white matter enormously swollen even as far back as the occipital region and softer than normal and somewhat jelly-like in appearance. The tumour was sharply defined (fig. 2).

It is submitted that in this case the rapidly fatal cerebral symptoms were directly referable to the condition of œdema of the right hemisphere, and not to the presence of the growth.

Symptoms which may Result from the Occurrence of Deposits Secondary to a Primary Cerebral Growth.

Secondary growths are very rarely met with in cerebral neoplasms. They occurred in two cases among this series. In both cases the primary tumour was situated in the neighbourhood of the fourth ventricle and in both the growth had burst through the ependyma or the pia and was in contact with the cerebro-spinal fluid. In one case, a secondary growth was found at the junction of the anterior and posterior roots of each of the spinal nerves, from the third cervical region to the lowest sacral region, a naked eye

appearance resulting of great enlargement of each posterior root ganglion. The filum terminale was the seat of many secondary growths, and its appearance resembled that of a string of beads. The symptoms referable to those secondary growth were severe shooting pains in all the limbs. Later, the knee-, wrist- and elbow-jerks were lost, and complete motor and sensory paralysis of the fifth and sixth cervical nerves on the right side occurred (fig. 3).

In the second case, a solitary secondary deposit was found upon the left first dorsal root. Right hemiplegia was present, and the patient lay in a half-unconscious state many weeks prior to death and no symptoms referable to the growth were detected.

In these two cases it was quite obvious that the secondary deposits had been planted by the cerebro-spinal fluid, and they afford important confirmatory evidence to the opinion that the main path of exit of the cerebro-spinal fluid from the intradural space is along the spinal nerve roots.

The nature of the neoplasms was that of fibro-sarcoma.

Symptoms which may Possibly Result from Posterior Degeneration.

The frequency with which degeneration of the posterior columns of the spinal cord occurs in association with intracranial tumours, no matter where these may be situated, has been pointed out by Dr. Batten and the writer, in this journal, vol. xxii., p. 473.

In some cases this degeneration is very marked and in three such cases among this series, shooting pains in the limbs, loss of the deep reflexes in the lower extremities, and ataxy of the legs, occurred, and were presumably referable to the posterior degeneration.

The Concealment of Localising Signs.

Many cases of intracranial tumour come under observation for the first time long after the onset of symptoms, and, in some of these cases, localising signs, which judging from

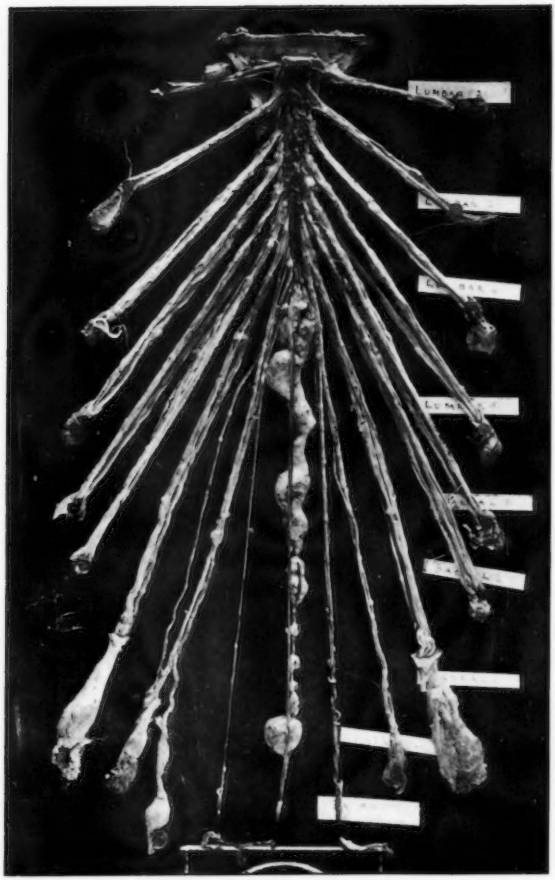

FIG. 3.

Secondary deposits in the filum terminale and root sheaths from a case of fibro-sarcoma of the cerebellum. Dr. F. E. Batten.

the situation of the lesion, must have been present in the early days, are no longer recognisable.

Hemianopia.

Two cases among this series came to the National Hospital presenting no localising signs and in which optic neuritis had produced complete blindness. Careful questioning, both of the patients and their relatives, revealed no indication that hemianopia had existed while sight remained.

In both cases, the tumour directly involved and apparently originated in the region of the cuneus and hemianopia must have been present early. But this, the only localising sign of a lesion in the posterior pole of the hemisphere, had been obliterated by the optic neuritis.

Cerebellar Signs.

While diagnosis should not be difficult in the early stages of cerebellar tumour, it may be very difficult in cases seen for the first time when the disease is of long standing. The signs of cerebellar disease may be called dynamic signs. It is necessary for their proper elicitation, that the patient be in the possession of his mental faculties, and of his power of voluntary movement. He must be able to sit up, to move his limbs, to stand, and to walk, and to attend intelligently to command.

In case of long standing, as a general result of intracranial pressure or of ventricular distension, mental deterioration, physical weakness, blindness from optic neuritis, and bilateral spastic paresis may conceal the cerebellar signs more or less completely, while the occurrence of hemiplegia, or of double hemiplegia from extension of the growth into the brain stem, or from pressure upon the brain stem, effectually conceals the cerebellar signs in the region of the paralysis.

The Disappearance of Local Signs.

The local signs which result from pressure and altered anatomical relations are sometimes temporary. It has been

A STUDY OF THE EMOTIONS.

BY W. H. B. STODDART, M.D., M.R.C.P.

(1) The Physical Basis of the Emotions.

In the following paper I shall endeavour to establish these two propositions:—

(1) That an emotion is a sensation-complex, its component sensations being entirely derived from a complex, usually involuntary, motor response to a percept or idea of some situation or incident.

(2) That the motor paths subserving the function of this involuntary response are those of the primitive nervous system, viz.:—the cortico-rubral system of fibres and the rubro-spinal tract.

There are some psychologists who would take exception to the word "entirely" in my first proposition, and who would say that organic sensations are merely superadded to a tone of feeling which is in some way attached to the idea of the situation. Professor Titchener, for example, writes in his "Outlines of Psychology": "It is essential, then, for the formation of an emotion, (1) that a train of ideas shall be interrupted by a vivid feeling; (2) that this feeling shall mirror a situation or incident in the outside world; and (3) that the feeling shall be *enriched*[1] by organic sensations, set up in the course of bodily adjustment to the incident."

Professor Titchener makes no attempt to explain the origin of this supposed initial feeling; but, as there are several psychologists who still share his view, it becomes necessary, in the first place, to re-state the case for the opposite side, which regards an emotion as a complex of organic sensations.

[1] The italics are mine.

already pointed out that indirect cranial nerve palsies may disappear in the course of time. Hemiparesis from pressure upon the pyramidal path may be similarly transient. As a tumour grows, the structural relations and directions of pressure are constantly changing and a region at one time pressed upon may subsequently be relieved. Possibly a structure may become used to the pressure as it were, and may regain its functional capacity, In two cases of glioma of the pons, however, the disappearance of a pathognomonic local sign was remarkable and difficult to explain.

Both cases, when admitted into Hospital, presented optic neuritis, bilateral spastic paresis of the limbs, much more marked in the legs and loss of lateral conjugate deviation. The last sign was indicative of interference with the sixth nucleus. Both patients lived many months and secondary hydrocephalus became manifest. Notwithstanding that the other signs became progressively more marked, the paralysis of lateral conjugate deviation became progressively less marked until some three months after admission it was no longer demonstrable. No other signs indicative of involvement of the cranial nerve nuclei appeared.

discover any stimuli which may be regarded as the cause of this superadded sensation.

The experimental results obtained by means of the plethysmograph, pneumograph, automatograph, &c., supply the evidence we seek. We find that in affective states, stimuli to muscular and circulatory sensations occur in all parts of the body; and it is to be inferred that these give rise to the superadded sensations which constitute the feelings of pleasantness and unpleasantness.

From the above considerations, therefore, we learn that feelings of pleasantness and unpleasantness are due to muscular and circulatory sensations which result from complicated movements evidently reflex in nature.

Now, the tone of feeling which attaches to a percept or idea is much more complex in nature than that which attaches to a simple sensation, and it has correspondingly a very much larger number of varieties. Inasmuch as the emotional tone differs with almost every percept, idea and concept of things, people, incidents and situations, which may occur, a satisfactory classification of the emotions is practically an impossibility. The feeling of attraction towards people and things may take the form of interest, familiarity, intimacy, reverence, or love. Repulsion may take the form of dislike, disgust, antipathy, contempt, repugnance, disdain, hatred, and perhaps anger. Ideas of welfare may be associated with feelings of satisfaction, gratitude, contentment, joy, hope, or anticipation; ideas of harm with feelings of sorrow, grief, dissatisfaction, resignation, despair, fright, or horror. And if the ideas are of the welfare or injury of others, we may have feelings of gratification, gladness, envy, jealousy, regret, care, or sympathy. To all these add the emotions of misery, effort, decision, defiance, pride, shame, mirth, and the emotional tones which characterise mental operations, such as recognition, comparison, discrimination, judgment and reasoning; and the list remains yet incomplete, because our language cannot take account of the numerous intermediate and mixed shades of feeling which may attach to any one of the above-named emotions.

The most elementary type of emotion is the feeling of pleasantness or unpleasantness which attaches to simple sensations. Introspection of these tones of feeling is very difficult, for reasons which will be understood later. For the present, it may be noted that attention to a tone of feeling necessitates inattention to the sensation by which it is aroused. Observations have been accordingly made on the physical concomitants of affection, and it has been shown by means of—

(1) *The plethysmograph*—That a positive tone of feeling (pleasantness) is accompanied by an increase of bodily volume (dilatation of arterioles), and a negative tone (unpleasantness) by a decrease.

(2) *The sphygmograph*—That a positive tone of feeling is accompanied by an increase of pulse frequency, a negative tone by a decrease.

(3) *The pneumograph*—That a positive tone is accompanied by an increase, a negative tone by a decrease of the depth of respiration.

(4) *The dynamometer*—That a positive tone is accompanied by an increase of muscular power, a negative tone by a decrease.

(5) *The automatograph* (a scientific form of planchette)—That a positive tone is accompanied by abduction of the arm, and a negative tone by adduction.

A tone of pleasantness or unpleasantness never arises without some or all of these physical accompaniments. They are the results of a general tendency on the part of the organism to reach out to that which is pleasant and useful and to withdraw from that which is unpleasant and harmful. There is little doubt that future research will discover many more movements associated with tones of feeling.

We are constantly using this phrase "tone of feeling" in discussing the nature of affection; and by retrospection, which differs but slightly from, and is in many cases the same thing as, introspection, we find that the phrase is well founded. The affective tone of pleasure or pain is a feeling or sensation superadded to the sensation which gives rise to it; and it becomes our duty to look round and see if we can

It has been pointed out by Professor James that an emotion consists of a number of sensations, and that these sensations are derived from the activities of certain muscles (voluntary and involuntary), and glands (sudorific, lacrymal, intestinal, &c.). The activities of involuntary muscles give rise to certain circulatory changes, such as increased or diminished frequency of the pulse, as well as to local flushings and pallors. And the more we investigate the matter, the more we become convinced that these sensations are the very essence of emotion. Let the reader conjure up some emotion, and note the various sensations he experiences therewith; then let him divest the emotional feeling of all these bodily sensations, and he will find that there is no part of the emotional feeling left. These various activities which give rise to the emotions are also responsible for the expression which indicates to others the nature of the particular emotion which is experienced.

According to this view, it is not the emotion which gives rise to the expression, but the expression which gives rise to the emotion. The question of the truth of this assertion has been appropriately referred to numerous actors: they have been asked whether they actually feel the various emotions they portray upon the stage. The best actors appear to be unanimous in the verdict that they actually feel the emotion they portray, especially when they are acting an emotional part well; and this is the reason why I have included the word "usually" in my first proposition. The experiences of the audience are in this connection no less interesting than those of the actor. When a member of the audience feels that he is being too much overcome by the sadness of the situation on the stage, he extends his trunk, assumes a smile, takes a deep breath, and surreptitiously wipes away the starting tear; by this means he dispels the emotion. And how often is an ill-timed merriment suppressed by assuming the expression, for example, of attention!

In this connection we now see why we are unable to attend to an emotion or affective tone. The particular combination of movements which constitutes the physical

basis of attention disarranges the particular combination which constitutes the physical basis of other tones of feeling.

In the emotions accompanying pleasant ideas there is an increase of muscular tone and power with a tendency to extension of the trunk and abduction of the arms, an increase of pulse frequency with general dilatation of the arterioles, and an increase of the frequency and depth of respiration. In the emotions accompanying unpleasant ideas the reverse bodily conditions obtain. This much we have learned from the study of affection. But, in addition to these physical signs, there are others in the various emotions, each complex giving rise to that expression which is characteristic of the particular emotion. In anger, there is contraction of the corrugatores superciliorum, fixation of the gaze, dilatation of the nostrils, tightening of the lips, grinding of the teeth, clenching of the fists, extension of the trunk, and flushing of the face. In disdain there is contraction of the levator labii superioris alæque nasi. In suspicion there is rapid lateral oscillation of the eyes. In dissent there is lateral nodding of the head, in assent there is antero-posterior nodding. If any of these expressions be well assumed, the corresponding emotion is experienced.

It has been shown by Darwin and others that all these apparently purposeless movements are in reality survivals of movements which have served some useful purpose in the past history of the race or of the individual. In anger, for example, the grinding of the teeth and clenching of the fists are survivals of similar movements performed upon portions of an enemy's flesh; the mouth being obstructed by such a portion of flesh, dilation of the nostrils becomes a necessity for breathing purposes. The gaze is fixed upon the enemy, and the contraction of the corrugators is necessary to protect the eyes from the sun during combat.

The contraction of the levator labii superioris alæque nasi in disdain is the uncovering of the canine tooth preparatory to biting the object of disdain. The oscillation of the eyes during suspicion is the search for anticipated danger. The lateral nodding of the head in dissent is a survival of the movement of refusing the proffered breast during infancy, and the movement of assent is that of accepting the breast.

The general conclusion is, then, that an emotion is derived *e motu*, out of motion, that it is a feeling compounded of sensations arising as a result of complex instinctive movements which are, as a rule, reflexly aroused by the situation in which the individual is placed.

In attempting to establish my second proposition, I leave out of consideration those emotions which are aroused by assuming the corresponding expression. This action is voluntary, and therefore has its physical basis in the motor area.

There is evidence to show that the thalamic region plays the most important *rôle* in the development of an emotion reflexly aroused. If a patient has a lesion of one optic thalamus, say the right, and you tell him a joke, he smiles on the same (right) side of the face only; the smile does not occur on the left side. That this paralysis is not due to a lesion of the cortex or pyramidal tract is shown by the fact that the two sides of the face act equally when he *assumes* a smile. If, on the other hand, the patient has a lesion of the right Rolandic area, he smiles equally on the two sides in response to a joke; but an *assumed* smile occurs on the right side only, volitional action being paralysed on the left side.

The observation of movements of expression occurring in the limbs is a more difficult matter in paralysed patients; the physician has to rely upon an opportunity of observing the hand when the patient yawns. In paralysis of the hand, due to some unilateral cortical lesion, the patient is unable to open the opposite hand voluntarily, but if he yawns, the hand opens slightly. If, however, a patient has a lesion in the region of one optic thalamus, he can open the opposite hand voluntarily, but it does not involuntarily open when he yawns. The conclusion to be drawn from these observations is that the tracts subserving the motor element of emotion cross to the opposite side of the cord.

Now, the only bundle which crosses from the mesencephalon to the opposite side of the cord is the rubro-spinal bundle of Monakow—that bundle which, as Held and

Probst, and subsequently Buzzard and Collier, have shown, arises on the ventral side of the red nucleus, decussates in Forel's crossway with the corresponding bundle of the opposite side, and is traceable in the region of the lateral tracts as far as the sacral region of the spinal cord. It connects the opposite nucleus ruber with the ventral horn of the cord.

I therefore submit that Monakow's bundle subserves the function of the motor element of emotion. The only other tract which here deserves consideration is the thalamo-spinal tract of Buzzard and Collier. This is an uncrossed tract taking its origin deep in the thalamus and traceable as far as the lowest cervical region. My data are not sufficient to include or exclude this tract from the function of emotional reaction.

I now have to consider the cortical portion of the system of motor neurons subserving the function of emotion. For this function a system of fibres is required to connect the cortex with the nucleus ruber, and such a system has been described by M. and Mme. Dejerine. The fibres take their origin in all parts of the cortex, but especially in the parietal lobe. They reach the thalamus just above the radiations of the internal geniculate body, enter into the constitution of the fillet, and reach the red nucleus at its antero-supero-external part. These fibres are to be regarded as the upper segment of the emotional motor system. Their intimate anatomical relationship with the thalamus easily accounts for the fact that that structure has hitherto been regarded as the physical basis of movements of expression.

There is no evidence for or against the view that the cortico-thalamic and thalamo-spinal fibres may play a subsidiary *rôle* as part of the physical basis of movements of expression.

It is of considerable interest that the cortico-rubro-spinal motor system is at least the main representative of the primitive motor tract, the tract by which in the lower vertebrates all motor impulses are transmitted. It has been demonstrated by Münzner and Wiener, Boyce and Warrington, Edinger and others, that the pyramidal system of

fibres does not exist in birds or in any of the lower vertebrates. In these animals the motor tract consists of cortico-thalamic and thalamo-spinal neurons only, the spinal fibres occupying the same relative position as the direct and crossed pyramidal system of mammals. In this connection it will also be remembered that in man the pyramidal tract is not myelinised until about the eighteenth month. Professor James has indicated the close relationship subsisting between emotions and instincts. Both are involuntary motor responses to sensations, percepts and ideas; and the only difference between them is that instincts bring the organism into more practical relation with the object of the idea, percept or sensation. Now, the lives of birds and lower vertebrates, and the life of the human infant until it is about eighteen months old, are practically little more than a mass of instinctive and emotional reactions; and it is not surprising to find that such reactions are, among the higher vertebrates, still dependent upon the functioning of the pristine nervous system.

The neural process which takes place when an emotion occurs is then as follows:—

Starting from the stage at which a sensation is registered in one of the projection areas, or a percept or idea formed in one of the association areas of the cortex, an impulse is transmitted to the red nucleus by way of the cortico-rubral fibres, thence to the large motor cells of the lowest level by way of Monakow's rubro-spinal (and presumably rubro-bulbar) fibres of the pristine motor system, and thence to the muscles of expression. Contraction of these muscles upon their spindles effects the transmission of muscle-sensations to the cortex by way of the ordinary sensory paths; and it is the particular combination of these sensations among themselves and with vaso-motor sensations which determines the given affective or emotional tone.

(2) The Pathology of the Emotions.

In this section I do not propose to deal with persistent states of depression or exaltation, but to consider morbid

modifications of the emotional reaction to percepts or ideas of situations and incidents in the outside world. In the insane such emotional reaction may be excessive or deficient, the cause of this excess or defect differing in the various states with which we have to deal.

In those conditions dependent upon progressive deterioration of the nervous system, such as general paralysis, alcoholic insanity and, perhaps, epileptic insanity, the emotional reaction is excessive; hemiplegics, too, are very emotional; the most unimpressive word or gesture often suffices to induce an attack of weeping or laughter. The same may be said of imbecility, a condition in which the nervous system is ill-developed, and of states of maniacal excitement. Again, paranoiacs and patients suffering from hallucinations are usually liable to outbursts of anger and other forms of emotion. We have also to consider those patients who suffer from morbid fears.

Deficient emotional reaction, on the other hand, characterises confusional and stuporose states, senility and all forms of secondary dementia, myxœdema, and cretinism.

(*a*) *Excess of Emotional Reaction.*

The doctrine is now well established that dissolution of the nervous system takes place in reverse order to its evolution; and it has been demonstrated that the last motor tract to develop in the history of the vertebrate nervous system is the pyramidal tract. It is in accordance with this doctrine that the first motor tract to suffer in such progressive degenerations of the nervous system as general paralysis, alcoholic insanity and epileptic insanity, is the pyramidal tract; and the consequence is that in these diseases, and also in hemiplegia, motor impulses tend more and more to be transmitted by way of more primitive motor tracts *viâ* the thalamus.

Now, these are the very tracts which normally subserve the function of the emotional reaction; and so it happens that patients suffering from the above diseases react emotionally to unimpressive stimuli: their main outlet is by way of emotion-arousing tracts.

I do not know whether the pyramidal tracts are especially ill-developed in imbeciles; but it is extremely probable that they are, for the patient is unable to walk properly in many cases. The nervous system as a whole is ill-developed, and it is to be supposed that the pyramidal tracts, which are normally myelinised late, are disproportionately affected. This being so, the above principle also applies to the excessive emotional reaction of imbeciles.

In states of maniacal excitement, the excessive tendency to emotional reaction is dependent upon a different set of conditions. I have elsewhere shown reason for the belief that mania is a state in which the neurons contain some irritating body or bodies; the neurons are consequently in a permanent state of excitability.

The result of this constant state of tension of the neurons in maniacal states is that minimal stimuli provoke nervous discharge and, in the case of motor neurons, induce contraction of muscles.

The application of this principle to the emotionality of maniacal patients is as follows:—A perceptual or ideational process occurs in one of the association-areas of the cortex; all the neurons in functional communication with this area, especially the cortico-rubral neurons, are discharged: in other words, discharge takes place into the emotional regions of the nervous system.

Dr. Wolseley Lewis has drawn my attention to the fact that insane epileptics, while free in the movement of their large proximal joints, are stiff and cramped in the smaller ones. He also observes that the epileptic shakes hands from the shoulder. In these particulars, the epileptic resembles the maniac; and, from the principles which I laid down in my paper "On Motor Symptoms of Mania and Melancholia" (*Lancet*, March 5th, 1904), it is to be concluded that the neurons of epileptics, as well as those of maniacs, contain some irritating body within their substance. The motor neurons of epileptics are therefore in the same "touch-off" condition as those of maniacs, and this affords us another explanation of the excessive tendency of epileptics to emotional reaction. Of course, it may be that the

patients to whom Dr. Lewis refers are cases of epileptic mania.

The emotional outbursts of paranoiacs and of patients suffering from hallucinations may be looked upon as being due to excessive perception, in contrast to those forms of diminution of emotional reaction which are due to "imperception" (*vide infra*). The laughing or weeping of a patient who has just experienced an illusion or hallucination takes place because the patient has perceived something which is not there; he has suffered from excess of perception.

Similarly, paranoiacs suffer from an excess of perception; their association of ideas is excessive, and they see hidden meanings in the most trivial incidents. A passer-by in the street blows his nose, and the paranoiac perceives the handkerchief as the cloak of a sneer or smile; the result is the emotional reaction we call anger.

Morbid fears are due to an abnormal tendency of the pristine nervous system to react to some particular percept. This tendency must, as a rule, be regarded as congenital, since it develops in people who are normally lacking in self-confidence. The tendency is, however, sometimes acquired; some illness leaves the patient devoid of self-confidence, or some unusual incident may initiate this morbid tendency.

(*b*) *Deficiency of the Emotional Reaction.*

If you tell a person a good joke, there are three possible causes for his not laughing at it: (1) He does not hear it, (2) he does not "see" it, and (3) he is preoccupied. These are the three causes of diminution of emotional reaction in the insane.

In confused and stuporose states the patient suffers from anæsthesia of characteristic distribution. In such cases perception is deficient because sensations are not satisfactorily served up to the ideational centres; and emotional reaction is deficient for the same reason that a deaf man does not laugh when you tell him a joke. It is also to be observed that in these patients there is a further reason for the loss of emotional feeling in that the muscular sense is defective, so

that the patient would not experience an emotional feeling even if a slight motor reaction should occur.

Emotional defect may be due to partial or complete imperception. In its typical form, imperception is a condition in which the patient is unable to recognise persons, objects and things; and it depends upon organic disease of the ideational areas. It also occurs to a greater or less extent in cases of secondary dementia, senility, myxœdema, and cretinism. In all these conditions there is corresponding deficiency of emotional reaction.

In some cases of this kind, emotional reaction occurs; but its character is inappropriate to the occasion. For example, it frequently happens that an advanced dement laughs on being told that some relative, once dear to him, is dead.

Absence or deficiency of emotional reaction also occurs in severe cases of melancholia. Professor Ribot mentions in his "Psychologie des Sentiments" some cases of melancholia in which there was complete absence of emotional reaction; but the name "anhedonia" which he has given this symptom and the descriptions of his cases indicate that he has not quite fully grasped its significance. Cases of severe melancholia suffer from an absence, not only of pleasurable, but also of painful, emotional reactions. Such patients experience no pleasure when they think of their home, wife and family; as a rule they tell us that they have lost all affection for their friends; and when it becomes our painful duty to inform a melancholiac of the death of his nearest and dearest relative, he commonly remarks, "I don't seem to feel it."

The cause of this lack of emotional reaction is not far to seek. I have frequently pointed out that the greater part of the muscular system of melancholiacs is rigidly fixed owing to partial paralysis of cortical neurons, and Dr. Craig's observations that the blood-pressure of melancholiacs is raised indicates some fixation of the involuntary arterial muscle-fibres. It is on account of this motor and vaso-motor fixation that the motor and vaso-motor changes essential to emotional reaction cannot take place.

Katatoniac stupor is also characterised by muscular rigidity. This rigidity differs from that of melancholia in that it affects the whole of the muscular system uniformly; whereas the rigidity of melancholia mainly affects the musculature of the spinal column and of the large proximal joints. Dr. Craig tells me that the blood-pressure is also raised in katatoniac stupor. The practical point is this, that in katatoniac stupor there is motor and vaso-motor fixation as in melancholia; and it is on account of this fixation that there is complete absence of emotional reaction in this condition.

Professor Kraepelin considers that there is an absence of emotional reaction in other forms of dementia precox. I have been unable to convince myself that this is the case, and, on the other hand, I am unable to discover any reason why there should be any lack of emotional response in most cases of simple dementia precox, hebephrenia or dementia paranoides.

The conclusions arrived at in this section may be summarised as follows: Excess or defect of emotional reaction may be dependent upon excess or defect of sensation, or upon excess or defect of perception. Excess of emotional reaction may also depend upon an abnormal tendency of motor impulses to be transmitted *viâ* the pristine emotion-arousing motor tracts of the nervous system; and defect of emotional reaction may further be due to fixation of emotion-arousing musculature.

SYMPTOMATOLOGY OF CEREBELLAR TUMOURS; A STUDY OF FORTY CASES.

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INTRODUCTION.

This paper is based upon the study of a series of forty cases of tumour which involved the cerebellum, either directly or indirectly by pressure, which we have per-

sonally observed during the past two and a half years in the wards of the National Hospital.

We here express our thanks to the Medical Staff for kind permission to use these cases.

The diagnosis was confirmed by autopsy or by operation in twenty-two cases, of which we append the clinical details.

We have rigidly excluded cases in which the disease extended into other parts, or in which conditions were present which could complicate the symptom-complex. This series is divisible into two classes: (*a*) Cases in which the tumour was situated within and limited to the substance of the cerebellum; (*b*) and those in which the growth was extra-cerebellar. By extra-cerebellar tumours we mean those which lie in the posterior cranial fossa in the angle between the pons varolii and the cerebellum, compressing but not directly invading either. Such growths may arise either in connection with the ventral surface of the cerebellum, in which case they are generally gliomata and occasionally cystic, or from the adjacent cranial nerves, when they are generally fibromyxomata. These growths are circumscribed, encapsulated tumours, and easily separable from surrounding tissues. Plates I. and II. represent photograph from a typical case, and illustrate the relationship of these growths to their surrounding structures. Despite the degree of compression which the pons suffers in such cases, it is remarkable how little the symptoms resemble those of intrapontine growth. The pressure is chiefly exerted on the middle cerebellar peduncle.

For a fuller study of the symptomatology we avail ourselves of eighteen cases in which the diagnosis has not received confirmation, but whose clinical picture was similar to those we record. This we can do with little hesitation, relying on the unvarying accuracy of the diagnosis in that series.

Finally, having had the opportunity of observing thirteen cases in which the disease was removed by operation, which entailed large destructive lesions, we are in a position to discuss the positive and negative symptoms of cerebellar

disease in relation to the results of experimental research in animals.

General Symptoms.

Headache is an early and almost constant symptom of both extra- and intra-cerebellar neoplasms. Its character is practically identical with that associated with other intracranial growths, but its situation is often suggestive of the site of the disease. In intra-cerebellar tumours the headache is generally most intense in the occipital region, and is frequently limited to this area for long periods, and in this respect is unlike the pain complained of in any cases of supratentorial neoplasms which we have observed. The pain may often radiate down the back of the neck to between the shoulders, and indeed the patient may refer it only to this region and show a great reluctance to moving the head owing to the discomfort it causes. The next most frequent site of headache is frontal or retro-ocular, but though this may be its initial situation it seldom remains limited to this region throughout the whole course of the illness. The frontal headache was never limited to one side in our cases. In extra-cerebellar tumours the pain is almost constantly occipital, and may radiate down the neck. It may not be so marked a symptom as in intracerebellar tumours, and, indeed, in some of our cases it was rarely complained of.

In contrast to the pain associated with intracranial neoplasms in other sites the headache is, as a rule, extremely severe and remarkably constant. We have found little in favour of Luciani's assertion, that it is due to direct irritation of the cerebellar tissue or of Purves Stewart's assumption that its occasional frontal seat is due to radiation of stimuli along the fronto-pontine tracts.

Vomiting is an almost invariable symptom during some stage of cerebellar and extra-cerebellar tumours. In the former especially it is apt to be very frequent and severe, but its character is identical with that which accompanies intracranial growths seated above the tentorium. When severe it is always associated with headache.

Optic Neuritis is in our experience a constant accompaniment of intra-cerebellar neoplasms. It seems to be one of the earliest signs, to be remarkably intense, and often out of proportion to the general symptoms. Its onset is as a rule acute, so much so that vision may be considerably impaired early in the illness. Attacks of transient blindness may occur during any stage of the illness, and often herald the approach of permanent loss of vision. In the majority of the cases the intensity of the neuritis and the degree of swelling of the papilla have been greater on the side of the growth and the permanent blindness due to consecutive atrophy has resulted in that eye first. In extra-cerebellar growths the neuritis is generally early and severe, but it may be late in appearing, and in one of our cases (No. 14) the patient died before any swelling developed. The degree of swelling was independent of the side of the tumour.

The general character of the neuritis is similar to that met with in other intracranial neoplasms.

Vertigo and Auditory Symptoms.

Vertigo is one of the most constant symptoms of extra- and intra-cerebellar tumours, and it is specially frequent in the former. Owing to its transient nature and the accompanying mental confusion it is often difficult to get a detailed or definite description of it from the patient. It may occur in two forms. In the one there is a definite sense of movement of self or of surrounding objects; in the other it is an indefinite sensation generally described as a "giddy feeling."

The subjective phenomena of definite vertigo need careful description. In both intra- and extra-cerebellar tumours the sense of displacement of external objects in front of the patient is from the side of the lesion to the opposite side. Thus if the tumour be on the left side the apparent movement of objects is from left to right.

In intra-cerebellar tumours the subjective rotation of self is

always from the side of the lesion to the healthy side, that is in the same direction as the apparent movement of external objects.

In extra-cerebellar tumours the subjective rotation of self is always from the healthy side towards the side of the lesion, that is in the opposite direction to that of the displacement of external objects.

The direction of the subjective movements was so constant in each class of case, that we are led to regard it as one of the most serviceable diagnostic points. These forms of vertigo were very pronounced in many cases after the tumours had been removed by operation. The patient may complain of a sense of movement of self or of external objects and at the same time tend to roll over in bed, in extra-cerebellar cases towards the side of the disease, in intra-cerebellar cases away from the side of the disease. These forced movements, which are not invariably associated with subjective movement, *i.e.*, vertigo, will be discussed more fully later.

In one case (No. 18) of extra-cerebellar tumour there was also an apparent vertical elevation of objects in front of the patient without subjective movement of self.

The other form of vertigo which is met with in both intra- and extra- cerebellar lesions is quite indefinite. The patient suddenly becomes giddy, generally after some abrupt movement, his surroundings become dark, and there may be some numbness of the extremities or a feeling of utter weakness, associated with intense nausea and a tendency to fall backwards or towards the side of the lesion. Consciousness may be impaired, but is never quite lost. Objects may seem to swim or intermingle indiscriminately in front of the patient, and he may feel that without support he must fall in any one direction or at once collapse. These forms of vertigo have no localising value.

In a few cases of cerebellar lesion, both before and after operation, we have rapidly rotated the patients on their vertical axes in order to observe the vertigo which results. In normal individuals when the rotation is suddenly ceased the person for a short time feels that he is still moving in

the direction of the rotation, and that his surroundings are revolving round him in the opposite direction. The eyes are at first in full conjugate deviation to the side towards which the rotation took place, *i.e.*, to the left when the patient is rotated from right to left, and regain their normal position by slow recession towards the middle line, interrupted by forcible nystagmoid jerkings towards that side. In the cases examined it was constantly found that the giddiness and the sense of rotation of self is less intense when the rotation has been towards the affected side than from it, and the deviation of the eyes is less complete and the nystagmus towards the middle line less marked. It must, however, be emphasised, that the eighth nerve, or at least its cochlear division, was affected in most of the cases so examined.

Deafness.—In considering this symptom the two classes of cases have to be rigidly separated. In our cases of extra-cerebellar tumours there was invariably some deafness on the side of the disease. The loss of hearing varied in intensity from complete deafness to relative reduction of acuity, which was always equally pronounced to air and bone conduction, its character being that of nerve deafness. In the intra-cerebellar cases on the other hand there was rarely any considerable affection of hearing. In a few cases there was slight deafness on the side corresponding to the disease, but this was never so great as in the cases of extra-cerebellar neoplasm.

Subjective Auditory Sensations.—Tinnitus is a fairly frequent symptom in extra-cerebellar tumours. We met with it frequently, and it was always referred to the corresponding ear. It was variously described as "the ringing of bells," "buzzing," or a "constant blowing or swishing noise." In intra-cerebellar tumours tinnitus was rarely a prominent symptom, and when present was vaguely described as "noises in the head," and was not referred to either ear. In this it resembles the tinnitus which occurs with intracranial neoplasms in other situations, and is probably dependent on the general increase of intracranial pressure rather than on local causes. Such subjective auditory sensations are never organised or elaborate.

Cranial Nerve Symptoms.

Ocular Symptoms.—The majority of cases of extra- and intra-cerebellar tumours develop, during some stage of the illness, varying degrees of external rectus palsy on the side of the disease, but the resulting squint and diplopia are often transitory, though liable to recur. This paresis is as a rule greater in extra-cerebellar lesions than in intra-cerebellar, and though in the latter there may be affection of both external recti, it is always most evident on the side of the lesion. Bilateral external rectus paresis is especially met with in tumours of the middle lobe of the cerebellum which compress the floor of the fourth ventricle. The degree of weakness is in all cases very variable, but is never complete unless the tumour secondarily involves the pons varolii, or when an extra-cerebellar growth extends so far medialwards on the ventral surface of the pons as to directly compress the sixth nerves.

Cerebellar lesions do not appear to have *per se* any influence on the pupils. The size of the latter depends upon the degree of impairment of vision due to the optic neuritis or secondary atrophy, but when there is secondary hydrocephalus the pupils may be very large, though reacting briskly to light and accommodation. Weakness of the muscles supplied by the third or fourth nerves never results directly from cerebellar growths. In one case, unconfirmed by autopsy, there was weakness of the superior oblique, probably due to direct compression of the fourth nerve by a tumour, which lay in the anterior portion of the cerebellum.

Another ocular phenomenon frequently seen, though never in complete degree, is some weakness of conjugate lateral movement towards the side of the lesion. It is met with both in tumours external to the cerebellum and in those of the lateral lobes, but is generally greater in the former class.

This weakness may be manifested merely by an inability to sustain lateral deviation of the eyes, or it may be that greater effort is required to move the eyes to that side. In other patients, especially those blind from secondary optic

atrophy, the eyes are often, while at rest, constantly deviated to the side opposite the lesion. Associated with this weakness of the conjugate movement of the eyes to the side of the lesion there may be relatively greater weakness of the external rectus of that side.

Skew deviation of the eyes is another ocular phenomenon which we have frequently observed after acute attacks or after operation. It consists of a downward and inward position of the eye on the side of the lesion, and of an outward and slightly upward position of the other eye. Its short duration suggests that it is due to an irritative condition rather than a destructive, though in one case it persisted for several weeks after operation, and in another, on which no operation had been performed, it was constant for considerable periods. (Plate II., fig. 4.)

Nystagmus.—This is a feature held in common by both classes of cases, and is one of the most valuable of the localising signs. It is almost invariably found in every case at some time or other. In the more chronic cases and when the symptoms have become latent it may be less marked and more variable. The typical nystagmus in cerebellar growths consists of slow deliberate jerking movements to the side of the lesion on looking in this direction, with gradual recession of the eyes towards the middle plane; in conjugate deviation of the eyes to the opposite side the movements are of smaller range, but occur in more rapid succession.

Though, as a rule, the nystagmus was conjugate, the movements of both eyes being equal in range and simultaneous, we occasionally observed a greater range of movement in the eye homolateral to the disease.

The nystagmus is always more marked on lateral than on vertical movements of the eyeballs. In a few cases on looking up there was irregular and coarse nystagmus often composed of rotatory movements of both eyeballs, but chiefly of the homolateral, upwards and to the side of the lesion.

Occasionally on convergence both eyes, but more frequently the homolateral alone, may jerk irregularly to the side of the lesion.

We have only observed spontaneous nystagmus in cases in which there was a marked tendency to conjugate deviation of the eyes, especially for a few days after operation. The movements were generally irregular, with a tendency to jerking of the eyes to the side of the disease and gradual recession towards their natural position.

N. Facialis.—Paresis of the facial nerve of the same side as the disease frequently occurs. It is of peripheral type, but in intra-cerebellar tumours is always slight and often variable and inconstant. As extra-cerebellar growths seem to have arisen from the flocculus or in connection with the auditory nerve, the degree of paresis in these cases has been as a rule greater and occasionally became complete, with reaction of degeneration in the later stages of the illness. In others, however, the facial paresis was very slight, or even absent when they first came under observation. In some cases of unilateral disease there may be facial palsy on the contralateral side, and it is then of the hemiplegic type owing to compression of the upper facial neurones in their passage through the pons. We have never observed facial weakness corresponding to the paresis of the homolateral limbs in cerebellar disease.

Twitching of the homolateral side of the face occasionally occurs in cases of extra-cerebellar tumour, presumably owing to direct irritation of the nerve.

N. Trigeminus.—Affection of either the motor or sensory divisions of the trigeminus occurs occasionally in intra-cerebellar tumours, more frequently and in greater degree in extra-cerebellar. In both classes the sensory root suffers more often than the motor. There may be only pain or paræsthesia (numbness, tingling, or a drawn feeling) on the corresponding side of the face, or definite sensory loss of variable degree. In the latter instance the corneal reflex may be diminished or lost and neurotrophic affection of the cornea may result. In these cases the sense of taste is frequently diminished or absent on the anterior two-thirds of the corresponding side of the tongue.

IX., X., XII. Cranial Nerves.—Affection of the functions of these cranial nerves is rarely if ever met with in intra-

cerebellar disease, but in some cases of extra-cerebellar tumour there was weakness of the same side of the palate, which on movement is drawn to the opposite side, and less frequently of the vocal cords and tongue. This was due, as confirmed in several autopsies, to direct pressure on these nerves by the growth, but only in the later stages of the illness did the weakness become pronounced. As a result of these palsies articulation became indistinct, slurred, or nasal, often scarcely intelligible, and even phonation was considerably impaired. The patients had increasing difficulty in swallowing, and as consciousness became clouded nasal feeding was required. Deviation of the tongue to the opposite side was occasionally observed in cases of extra-cerebellar tumour due to the pressure on the pons varolii being exerted on the central hypoglossal connections above their point of decussation. It was in these cases that some spastic weakness of the contralateral limbs existed.

Motor Symptoms.

In making observations on the motor system special attention has been paid to the following points: (1) The tone of the muscles; (2) the power of the various movements; (3) the co-ordination; (4) the attitude of the patient; (5) the gait. In analysing the cases it will be seen that in some of the intra-cerebellar cases the onset was recent and acute, while in others slow and gradual, and in many of the cases operated on there followed as a necessary sequence destruction of various portions of the cerebellum. Thus we have had the opportunity of observing the motor system: first, in acute intra-cerebellar growths; secondly, in chronic intra-cerebellar growths; and thirdly, of noting the immediate and late effects of extensive or more limited destructive lesions of various regions of the cerebellum which resulted from operative interference.

Paresis.—One of the most definite symptoms of unilateral cerebellar tumour is deficiency of power in the limbs of the same side. This hemiparesis is often considerable when compared with the power of the opposite limbs; in some

cases, however, it is absent. It may be said to be uniform as far as the distal and proximal muscles of the extremities are concerned. This muscular asthenia must be a direct effect of the cerebellar lesion ; as it is homolateral to the side of the disease, it cannot be ascribed to any effect, direct or indirect, of the tumour on the cortico-spinal system, and in cases which terminated fatally no degeneration of these fibres could be found on microscopical examination.

Further, the character of the weakness, the absence of any organic rigidity and the normal state of the superficial reflexes, are strongly against the existence of any interference with the functions of the pyramidal tracts.

The weakness is relatively greater in cases of acute onset, and especially after extensive destruction of cerebellar tissue entailed by operation. In the latter class of cases there is often a condition of general muscular asthenia, but there can never be any doubt as to the greater homolateral paresis. The contralateral limbs very quickly regain their full power, but their fellows may remain definitely paretic for a considerable period. As regards the distribution of the weakness, there is a very considerable paresis of the trunk muscles, especially of those of the vertebral column, and it would appear that this weakness is greater in the muscles of the homolateral side. We have been able to observe this only in certain cases.

Paresis of the trunk muscles was most definite for variable periods after operation. These patients were unable to sit up in bed without support for any length of time, they could not straighten their backs and fell limp in any direction. This seemed to be due to the inability of the spinal muscles to bear the weight of the body. In cases which recovered a definite degree of lordosis when standing persisted when most of the other symptoms had disappeared.

In cases of extra-cerebellar tumour there was seldom such definite paresis of the homolateral limbs as was observed in the intra-cerebellar growths, except after operations from which additional cerebellar lesions resulted. On the other hand, we frequently found evidence of spastic paresis of the contralateral limbs and accompanying reflex changes charac-

teristic of organic hemiplegia. This was undoubtedly due to the direct pressure of the tumour on the cortico-spinal system in the pons, and of this there was definite evidence in the fatal cases. This association of contralateral hemiparesis with cranial nerve palsies on the side of the lesion presents the clinical picture of crossed hemiplegia. In one or two advanced cases the pressure had been so great as to affect both pyramidal tracts.

Atonia.—Associated with the unilateral weakness there is often a defect of tone in the muscles of the homolateral limbs, so that during rest they assumed unwonted positions, and when handled are found to be limp and flaccid. The muscles may be soft and flabby to the touch. When such a limb is held at a proximal segment and shaken the distal segments flop about like the limb of a flail, apparently unrestricted by any active or passive tone of the muscles. The greater degrees of this hypotonicity are met with early in cases of acute onset, and especially after operations in which the cerebellum has been considerably damaged. In the latter cases, though chiefly homolateral, the opposite limbs may also be slightly hypotonic, but quickly recover.

The relation between this atonia due to cerebellar lesions and the state of the tendon reflexes is inconstant. While the hypotonia of tabes dorsalis and acute peripheral neuritis is always accompanied by loss of the tendon reflexes, the hypotonia of cerebellar lesions may be associated with definite increase of these reflexes, or the latter may be absent or diminished in various degrees.

For the first few days after operation when the deficiency in tone has been most marked, we have invariably found the tendon reflexes absent, but their rapid return did not correspond with any objective change in the tone of the muscles, and they even became exaggerated before any definite increase of tone occurred. This forces us to the conclusion that the state of the deep reflexes is not any measure of the muscle tone in such cases.

The irritability of the muscles to direct percussion was often more brisk on the homolateral than on the opposite side.

There is one phenomenon which we have repeatedly observed associated with, and apparently due to, hypotonia. When a person attempts to execute a movement against resistance with a normal limb, and the resistance is suddenly removed, the limb rapidly moves a short distance in the desired direction till abruptly checked, or it may recoil. In a spastic limb the amount of recoil is excessive; in functional cases there may be neither primary movement nor recoil. In homolateral cerebellar disease the range of movement is excessive, generally continued till it is no longer mechanically possible, and there is little or no recoil. This absence of recoil must be attributed to the inability of the antagonistic muscles to react in tone to the sudden strain to which they are exposed (reflex muscle tone).

The simplest way to test this phenomenon is to request the patient to flex the forearm as strongly as he can, the elbow being firmly supported and the movement effectually resisted by the observer grasping the wrist. The resistance is suddenly relaxed by releasing the wrist.

We have rarely noted any considerable change in the tone of the muscles of the homolateral limbs in cases of extra-cerebellar tumour. With evidence of pressure on the pyramidal tracts there may be varying degrees of hypertonicity of the muscles, especially of those of the contralateral limbs.

Ataxia. — Some degree of ataxy of the homolateral limbs is one of the classical signs of unilateral cerebellar affections. The defect consists in a deficient control of the various component muscular contractions by which a complex act is executed, and in a disturbance of the normal association of the action of muscles with that of their antagonists, which regulate or limit the act. It is, in fact, a dysmetria. The individual components are ill-judged, being generally in excess of the requirement and inaccurately associated in time, the result being that the purposed act is only attained after several deviations from the direct line of movement. A special character of the ataxy is that it is directly due to this lack of accurate co-operation and associa-

tion between the individual muscular contractions and not to impairment of muscular sense or of the sense of position. In other words, its origin is central, not peripheral. The patient is unable to check the deviations though fully conscious of his error. Unlike the ataxy of tabes dorsalis the deviations are not increased when the patient is deprived of the aid of vision. The ataxy appears only in active movement of the limb, does not increase towards the completion of the action, and, finally, the limb remains steady when its object is attained. In these characters it is the counterpart of the irregularity of movement styled intention-tremor, met with in disseminated sclerosis. Cerebellar ataxy is within wide limits more or less characteristic, and when the type is once recognised there should rarely be any difficulty in distinguishing it from other irregularities of movement. It is most evident in cases of acute onset or during acute exacerbations of the other symptoms, and especially after operative destruction of the cerebellum.

In cases of chronic course or where the lesion has become latent the ataxy is, as a rule, less definite. This is especially so when the condition has become complicated by hydrocephalus; then it may be less typical and approximate to the intention-tremor type characteristic of disseminated sclerosis. This seems to occur most frequently in tumours involving the vermis. In disease of one lateral lobe the inco-ordination is typically present only in the homolateral limbs, but not infrequently there is, in addition, some degree of ataxy in the opposite limbs, though always less in degree. When the tumour is in the vermis, or extends into both lateral lobes, the inco-ordination is bilateral, but greater in the limbs of the side on which the lateral lobe is more affected, or it may be more pronounced on one or other side as the effect of the tumour varies. The inco-ordination affects the arm more than the leg. The reeling gait would seem to suggest that it is greater in the lower extremities, yet, on examination, it is found that this is due in great measure to the irregular action of the trunk muscles. We have often noted when the patient stands erect that an unnatural degree of alternate contraction of the erectores spinæ of the two

sides occurs as he attempts to maintain or attain his proper balance. Patients who are so unsteady on their feet that they can scarcely walk, present a comparatively slight degree of inco-ordination in carrying out movements as they lie in bed.

We have not been able to make out any constant difference between the ataxy which results from cerebellar disease, and that met with in extra-cerebellar tumours.

Some slight degree of inco-ordination generally persists for many months after the disease has been removed by operation, and may be one of the last signs to disappear.

We have carefully investigated in a number of our cases the sign described by Babinski as pathognomonic of cerebellar disease, to which he has given the name "diadocokinesia." It consists in the inability of the patient to accurately perform rapid alternate movements with the homolateral limb, though the individual movements are easily possible. It is tested by requesting the patient to pronate and supinate the forearm in rapid succession; this can be quite naturally executed by the contralateral limb, but the homolateral arm is moved slowly, awkwardly and irregularly. This phenomenon seems to forcibly confirm our analysis of cerebellar ataxy—that it is a dysmetria or defective control of muscular action, with inaccurate combination of the component muscular contractions.

It seems to us that this inability to rapidly repeat movements is dependent upon the defective cooperation of the muscles and their antagonists, due in part, at least, to the diminution of the reflex muscle tone described, and to the consequent failure of the antagonists to control the primary movement owing to their lack of tone.

We are not, however, in agreement with Babinski that it is pathognomonic of cerebellar disease, nor have we found it constantly present in all cases of cerebellar lesions where we have looked for it. It seems to occur equally in cerebellar and extra-cerebellar tumours.

Attitude.—In many cases of unilateral cerebellar and extra-cerebellar tumours the head is held in a more or less characteristic attitude. When the patient sits or stands it

is slightly flexed to the side of the lesion and rotated so that the chin is directed towards the opposite shoulder and the occiput approximated to the point of the homolateral shoulder. Though very characteristic, this attitude is neither constant nor pathognomonic. Occasionally in cerebellar growths the head is held in the reverse position, and this "cerebellar attitude" of the head is met with in cases of pontine, mid- and, more rarely, of fore- brain neoplasms. It cannot, therefore, of itself be used as a positive sign of cerebellar disease or as a definite aid in its localisation.

In some cases of tumour of the middle lobe there may be a tendency to retraction of the head; in others the head may be flexed and bent forward until the chin rests upon the chest, especially when the patients walk. We have also observed in a large proportion of the cases lordosis in the dorso-lumbar region when the patient assumes the erect attitude. Some patients, however, stand with their body rather inclined forwards. This seems to be most common in neoplasms of the vermis, probably in those cases where the disease is chiefly situated in its anterior part, and we have noted it specially after operation. Two of the patients when first placed upon their feet assumed a sprawling attitude, which was probably due in part to the general asthenia. We have not seen any tendency to pleurotonus or lateral curvature of the spinal column, though in unilateral disease patients are often inclined to bend over to the side of the lesion.

Involuntary Movements.—In unilateral cerebellar tumours which are not complicated by hydrocephalus, the homolateral arm is held remarkably steady when extended horizontally in front of the patient. The absence of any tremor, vibratory muscular contraction or spasm and the ability to maintain one position for considerable periods, is striking in a limb whose volitional movements are inco-ordinate, and emphasises the purely kinetic character of the ataxy. The contra-lateral arm may be unsteady or tremulous when held in the same position, but not as a rule to an unnatural degree.

In extra-cerebellar tumours, on the other hand, there is

often irregular tremor in the horizontally extended arms, as a rule equally well marked in the limbs of either side. This tremulousness is in striking contrast to the unusual steadiness noticed in the homolateral arm in cases of unilateral cerebellar disease.

In a few cases there was for variable periods after operation some tremulousness of various parts of the body when unsupported, but as a rule it did not persist for long. It was most frequently observed in the head and trunk; when the patient sat up or was placed in the erect position there were constant nodding or bowing movements of the head and body, and at times lateral oscillations. There was similar spontaneous movement of the limbs, especially of the homolateral, when under analogous conditions. The muscular contractions from which they resulted seemed to occur only in the relaxed muscles, as the limbs remained fairly steady when rigidly extended in front of the patient. This condition seems to be identical with the astasia observed in animals after destructive lesions of the cerebellum.

Gait.—The gait of cerebellar disease has been accurately described as drunken, staggering, or reeling, but further analysis of its characteristics is necessary before it can be used as an aid in localisation of the lesion. There appear to be two distinct components which characterise the gait peculiar to unilateral cerebellar disease. (1) A tendency to stagger and stumble to the side of the lesion; (2) a tendency to deviate from the desired direction towards the side of the lesion. The stumbling is more or less characteristic; the patient when walking suddenly totters as though to save himself from falling, as if he were drawn by an unseen influence towards the side of the disease, but, as a rule, he quickly recovers his balance and direction. He may occasionally stumble to the opposite side, but he does so less frequently and to a less extent. This stumbling is rarely of such a kind as to suggest that it is due to weakness or giving way of the homolateral leg.

The deviation from the desired line of progression is not less characteristic, but often requires to be carefully analysed. In acute unilateral cerebellar lesions there is a

constant tendency to deviate to the side of the lesion, so that, were it not corrected, the patient would describe a definite circus movement to this side, *i.e.*, with the homolateral shoulder towards the centre of the circle. But the patient is fully conscious of this tendency and almost invariably attempts to correct it, and when the symptoms have persisted for some time he may do so almost automatically. This he does by rotating the trunk so that the homolateral shoulder is held in front of, and generally higher than, its fellow, the transverse axis of the body being thus directed from the line of progression towards the side opposite to the lesion. Thus he substitutes for the potential angle of deviation towards the side of the lesion a virtual angle of deviation towards the opposite side, by means of a complementary rotation of his trunk in that direction through an equal angle.

When the disease has lasted for any considerable time, especially when the patient is daily up and about, there may even be an over-correction of the original deviation, so that he tends from the direct line towards the healthy side. This is seen on those days when the patient's gait is better than usual. If this over-correction is not fully recognised it may lead to misinterpretation and wrong localisation. Other patients may attempt to correct the tendency to deviation and stumbling by throwing the weight of their body as far as possible on the contralateral foot by leaning to that side, but this is of rare occurrence. The actual tendency to deviation from the direct line of progression towards the side of the lesion seems to be the essential condition. In acute cases it has always been very definite and was often recognised and spontaneously described by the patients as a sensation of compulsion or traction in that direction. For instance, a patient with left extra-cerebellar tumour, was walking very unsteadily when first examined, and spontaneously remarked, "I know I am always going to the left, but I can't help it; I feel as if something were pulling me over." Next day his gait was much better, and he deviated to the right, which he thus explained, "I am always inclined to go to the left and try to overcome it

by going to the right, and sometimes when I am steadier I go too much to the right."

There is no constancy in the direction in which the patients, collectively or individually, turn round; as a rule they stop for a moment, and then turn slowly and awkwardly, more often perhaps round the side of the lesion.

In many cases, and, indeed, most commonly, the patient holds the homolateral leg more abducted than its fellow, and also rotated at the hip, so that the foot and toes point outwards. In this way he broadens his base. We have also noted frequently a definite ataxy or awkwardness of movement of the leg of the affected side as the patient walks, and he can always stand more steadily on the leg opposite to the side of lesion than on the homolateral. When the feet are placed together, the tendency is generally to fall backwards and to the side of the lesion.

In mesial tumours, or those involving both lateral lobes, the gait is more truly drunken or ataxic, and the patient stumbles and reels from side to side. In these cases there is often a tendency to fall backwards or forwards rather than to either side, and in one of our cases at least there was definite retropulsion; the patient when placed on his feet constantly stumbled backwards, as though pushed, at the same time deviating to the side most affected by the lesion, so that when supported he described a circle with the homolateral shoulder towards its centre.

In extra-cerebellar tumours which exert pressure on the pyramidal tracts in the pons the gait may be complicated by definite spastic paresis of the opposite leg, so that the patient may stand better on the homolateral leg than on the opposite one, and may stumble on the spastic limb.

In all cerebellar lesions the subjective uncertainty and unsteadiness seem to be often in excess of the objective disturbance. After operation the patient may be unable to sit up or walk for some time. When improvement sets in, he walks in the manner described above but with all the features more marked. Recovery is often rapid, but it may be some months before he can walk quite naturally. In several cases the lordosis and the stumbling to the side of the lesion were the last signs to disappear.

Sensory Symptoms.

Alteration of cutaneous sensibility never occurs in cases of cerebellar disease except when the tumour involves the pons, either directly or indirectly, by pressure, or causes secondary softening. Even in extensive lesions of the cerebellum, as where one lateral lobe has been practically destroyed, there has been no appreciable change in any form of sensation. The sense of position of the limbs, stereognosis, and the correct appreciation of weight, are also never impaired. Some anæsthesia over the distribution of the trigeminal nerve, however, may result from direct pressure on its sensory root, especially in cases of extra-cerebellar growths.

Cranium.

In the younger patients there may be definite bulging of the skull in the occipital region over the seat of the tumour. We have been unable to observe this in any of our adult cases, though some thinning of the skull was occasionally found on operating. In the majority of the cases there has been considerable tenderness on pressure over the occipital region, generally greater on the side of the disease. Auscultation and percussion of the skull never yielded positive signs.

Mental Condition.

Cerebellar growths do not seem to have any effect on the psychical functions, *per se*, though any acute increase of intracranial pressure may cause a drowsy, or stuporose state, as a rule of brief duration.

Reflexes.

Tendon reflexes.—The variability of these reflexes is one of the most striking signs of cerebellar lesions. In some cases they are increased, while in others they are diminished or lost, but any one case may present both extremes within short periods. The knee and ankle jerks illustrate this fact best, not only because they vary more, but the degree of change is most noticeable in them. In many cases where the tendon reflexes of the upper extremities have remained natural or brisk throughout, those of the knee and ankle were at different

times diminished or absent. In unilateral cerebellar tumours diminution on the side of the lesion is perhaps the most common condition, though an increase on both sides, or on the side of the lesion, is often met with. We have never obtained ankle clonus in such cases. In tumours involving the middle lobe there is generally an increase in the deep reflexes on both sides, and this condition is rarely absent when secondary hydrocephalus has developed.

In extra-cerebellar cases the tendon reflexes are rarely diminished; in some they remain natural, but as a rule they are increased, especially on the contralateral side. We have never observed any relative difference between the tendon reflexes of the upper and lower extremities. Ankle clonus is found fairly frequently associated with spasticity of the limbs opposite to the lesion, and in a few cases it may be present on both sides.

In all the cases which recovered we have noted absence of the tendon reflexes for some days following operation; they, however, quickly reappeared, and always on the contralateral side first. Their loss coincided with the extreme muscular asthenia and atonia to which we have already referred, but their reappearance and subsequent increase was not synchronous with any appreciable improvement in the muscular tone.

In some cases ankle clonus developed in the homolateral side while the asthenia and atonia of the muscles still persisted. This ankle clonus was scarcely identical with that met with in spastic states; it was irregular, intermittent, and of short duration, and was unaccompanied by any change from the flexor type of plantar reflex, or other evidence of interference with the pyramidal system. The presence of ankle clonus in these conditions is noteworthy, as attention has not yet been drawn to its occurrence.

Superficial reflexes.—These are usually natural in character, except in those cases in which there is pressure on the cortico-spinal system. When hydrocephalus has developed they may be diminished or absent on both sides, and when one pyramidal system has been pressed on they are diminished on the opposite side.

Plantar reflex.—We have never obtained an extensor plantar response in any case of uncomplicated intra-cerebellar lesion.

In extra-cerebellar tumours, on the other hand, where there has been pressure on the pons, an extensor plantar response may be found on the opposite or on both sides. Despite the large amount of compression these tumours exert on the pons a contralateral extensor response on plantar stimulation seems to occur only in the later stages, and in some cases has been absent throughout their whole clinical course.

Fits and Spasms.

In cerebellar cases a history of "fits" was occasionally obtained from the patients or their friends, but on closer questioning it generally appeared that these were attacks of vertigo of the severer form with clouding of consciousness.

We have, however, observed in one case a long series of seizures of peculiar nature.

In case 9, in which a large tumour lay in the under surface of the vermis, the operation by which its removal was attempted resulted in considerable hæmorrhage into the substance of the left cerebellar hemisphere, chiefly in the region of the dentate nucleus. This patient had a series of tonic spasms all more or less alike. The first occurred immediately after the operation while he still lay on the table recovering from the anæsthetic (chloroform). The face was unaffected by spasm, and respiration, which was slow and deep, was also undisturbed. Both arms were rigid and resistent to passive movement, the left more so than the right; they were adducted to the sides, extended at the elbows and wrists, while the fingers were flexed at all joints and covered the thumb. The left arm was rotated strongly inwards at the shoulder and the fore-arm pronated so that the ulnar border of the hand faced forwards, while the right arm was rotated slightly outwards and the fore-arm supinated till the palm of the hand came to look forwards. Both lower extremities were rigidly extended at hip, knee, and ankle, the rigidity being greater in the left. The left limb was rotated inwards and the foot inverted, the right rotated outwards with the foot everted. The position of the back was not fully observed in this fit, but there was a tendency to roll over

to his right side. The spasm was purely tonic in all limbs, and uniformly maintained for about three minutes.

At the onset of the fit the eyes were in conjugate deviation to the right, but in its later stages they rolled slowly from side to side, their axes, however, not remaining parallel, as the eye of the side towards which the movement took place started first. There followed, immediately after its cessation, two similar fits in close succession.

The child never fully regained consciousness, though the corneal reflexes were present, and he occasionally cried out. After an interval of about an hour and a half the fits recurred.

As in the fit described, the onset was sudden without local commencement, and there were no clonic movements. The face was not affected, the head was retracted and rotated to the right, the back arched and laterally curved so as to be concave to the left, and the body rotated on the vertebral column to the right, so that it seemed as though he were rolling over to this side as he lay on his back. The position of the arms was as above described, except that in the severer fits there was flexion of the wrists. The rotation of the limbs, inwards at the left hip and shoulder, outwards at the corresponding joints of the opposite side, as well as the pronation of the left fore-arm and supination of the right, became more marked. When the spasm was strong, the adduction of the left limbs was pronounced and the right were abducted from the side.

The eyes behaved as in the first fit, that is, during the early stages both were in extreme deviation to the right, while later, as the spasm began to pass off, they rolled slowly from side to side, the eye of the side towards which the deviation was occurring beginning the movement and retaining its lead till it reached the outer canthus, though not to the full extent, as the other eye followed at a quicker rate. When both eyes had reached the limit of range to the one side, they commenced to move in the opposite direction in precisely the same manner. The movement of each eye was slow and uniform, a single excursion occupied about ten seconds.

The patient only survived the operation about nine hours, but during this time he had a very large number of seizures, which were carefully observed. They were all of the same type as those described, but the retraction of the head, the opisthotonus and the lateral curvature of the spine with the concavity to the left and its rotation to the right became more marked. In these later fits, adduction of the left limbs and abduction of the right were constantly observed.

Each fit passed off gradually, the tonic spasm slowly relaxing, first in the right limbs. The limbs were remarkably flaccid and atonic in the intervals between the seizures.

Summarising from these descriptions it will be seen that each fit consisted in rotation of the head, trunk and limbs on their longitudinal axes from the side of the lesion to the healthy side, and deviation of the eyes in the same direction. The homolateral limbs were also adducted, and the contralateral abducted. There were no clonic movements at any stage of the many seizures observed. Their onset was abrupt, but the relaxation of the spasm gradual. In many of the fits the tonic spasm was greatest in the muscles of the trunk, and in all of them it was more pronounced in the homolateral limbs than in the opposite.

We have also had the opportunity of observing a few seizures which affected the trunk and limbs in an exactly similar manner in the case of a girl where a large tumour infiltrated the pons and one lateral lobe of the cerebellum.

It is probable that these phenomena must be ascribed to an irritative lesion of the cerebellum. Those in the case we report were probably due to the recent hæmorrhage, and their short duration and frequent recurrence suggest that they were irritative phenomena. We have never observed similar seizures in forebrain lesions, and in the fact that they were purely tonic they seem to be the counterpart of the clonic spasms which result from cortical discharge.

Dr. Hughlings Jackson has described a variety of tonic cerebellar fits which occur in cases of tumours of the vermis. In these there is retraction of the head, arching of the back with flexion and supination of the elbows, rigid extension of the legs and pointing of the toes. We have never observed such, but it is conceivable that they are due to a compound of discharges similar to those we described from each lateral lobe of the cerebellum, dependent upon disease of the middle lobe.

A few of our other patients have given accounts of jerking movements of the homolateral arm, often associated with attacks of vertigo. In one case of extra-cerebellar tumour (No. 18) in which such involuntary movements were observed, they preceded the vertigo and consisted of an isolated jerk, or a short series of jerks, of sudden shock-like

character of any part of the homolateral arm, lasting over a period of two to three minutes. The movements were irregular in distribution and sequence, and according to the patient occasionally affected the contralateral arm simultaneously, but always in slighter degree.

Differential Diagnosis.

It seems to us that when due attention is paid to these symptoms and physical signs the accurate diagnosis and localisation of the majority of uncomplicated cerebellar neoplasms is possible. On the one hand, it may be asserted, from the examination of the records of a great number of cases observed in past years in the National Hospital, that cerebellar tumours rarely, perhaps never, run a latent course or fail to produce clinical symptoms, and, on the other, this typical symptomatology is never met with in other conditions unassociated with other signs.

In considering the question of diagnosis, supratentorial tumours may, in the first instance, be placed in contradistinction to those situated in the posterior fossa and can, as a rule, be easily distinguished from them. Within the latter class are those seated in the substance of the brain-stem, those within the cerebellum, and those which lie without and only affect these parts by pressure. It is the differential diagnosis between these three varieties which we propose to discuss.

(*a*) *Between Cerebellar and Extra-cerebellar Tumours.*

As a rule the cardinal symptoms of intracranial neoplasm —headache, vomiting and optic neuritis—appear early and in pronounced degree in intra-cerebellar growths, while in cases of extra-cerebellar tumour any or all of them may first occur late in the history of the case. In the former class the functions of the sixth, seventh and eighth cranial nerves may be impaired, but always in slight degree. In extra-cerebellar tumours the affection of these nerves has been invariable and often considerable. The position of the

growth in each case will naturally determine which of them will suffer most, but in those we have observed, marked or complete deafness resulted early, and there was generally definite affection of the facial nerve, which might even advance to complete palsy, with electrical reaction of degeneration.

Evidence of pressure on the motor or sensory roots of the trigeminus is rarely met with in cerebellar tumours, but commonly occurs, often in considerable degree, in the extra-cerebellar group. Weakness of the palate or vocal cords, with difficulty in swallowing and affection of phonation or articulation, seems to belong to the symptom-complex of extra-cerebellar tumours, as we have never met with these symptoms in uncomplicated cerebellar disease.

Marked affection of the functions of the cranial nerves, and especially disproportionate palsy of one, is, other things being equal, strongly in favour of the diagnosis of extra-cerebellar tumours.

Vertigo when present may be a useful aid in localisation. In both classes the movement of objects in front of the patient was always, in our cases, from the side of the lesion to the opposite side, but while in intra-cerebellar lesions the subjective rotation of self is in the same direction, in cases of extra-cerebellar tumours it has always been in the reverse.

In extra-cerebellar tumours the homolateral paresis and hypotonia is often absent or is less marked than in cerebellar lesions, but we have noticed no difference in the degree or quality of the ataxy or in the character of the gait. As above-mentioned, the hands are generally held remarkably steady in intra-cerebellar lesions, especially that of the homolateral side, but in extra-cerebellar we have frequently found considerable coarse tremor of the horizontally extended limbs, which, in some cases, became so accentuated on volitional movement as to resemble intention-tremor.

The association of cerebellar symptoms with crossed hemiplegia of even the slightest degree is strongly suggestive of an extra-cerebellar tumour compressing the pons. In our experience, an extensor plantar response absolutely excludes uncomplicated cerebellar disease.

Tumours situated in the middle lobe of the cerebellum can rarely produce such symptoms as to necessitate the serious consideration of their differentiation from lateral recess growths.

(*b*) *From intrapontine tumours.*—The symptomatology of intrapontine tumours varies immensely according to their situation and nature. In soft-infiltrating gliomata, such as form the pathological condition formerly known as "hypertrophy of the pons," the cranial nerves may be remarkably spared, while if the growth is firm and circumscribed it destroys all neighbouring structures indiscriminately, and produces quite a different clinical picture. It is possible to confuse the symptoms of the first variety with those of intra-cerebellar lesions, while those of the second variety tend to resemble those of extra-cerebellar tumours. The almost invariable absence of optic neuritis, the less intense headache and vomiting, and the early appearance of evidence of involvement of the cortico-spinal system in pontine tumours generally allows a correct diagnosis.

The inco-ordination which results from intrapontine lesions may resemble that seen in cerebellar tumours, but it is, as a rule, less definite in quality and situation. Cranial nerve symptoms, when present, are often bilateral and more general than is the rule, even in extra-cerebellar tumours, when one or two nerves involved in the growth are affected out of all proportion to the others. Such complete and permanent palsy of lateral conjugate deviation of the eyes as very frequently results from intrapontine growths, never never occurs in cerebellar or extracerebellar tumours.

Tumours situated further cerebralwards in the quadrigeminal or midbrain regions may produce some cerebellar symptoms, but the impairment of function of the cranial nerves of this level, with or without crossed hemi-paresis, and, perhaps, associated with hemi-tremor or hemi-anæsthesia, prevents, as a rule, any confusion.

The following table shows the chief points to be considered in the differential diagnosis of these three varieties of cases.

Symptoms and Signs.	Lateral Cerebellar Tumours.	Extra Cerebellar Tumours.	Intrapontine Tumours.
Optic neuritis ...	Early and intense ...	Variable	Often absent or late.
Vertigo	Subjective rotation of self from the side of the lesion	Subjective rotation of self to the side of the lesion	Indefinite.
Cranial nerves V.	Rarely affected	Often affected... ...	Affection of these nerves often bilateral. Paresis may be supranuclear or nuclear, and grouped according to nuclear arrangement. Paralysis of a nerve on one side and of an adjacent or distant nerve on the opposite side. Permanent paralysis of conjugate deviation of the eyes.
,, ,, VI.	Weakness of conjugate deviation to side of lesion. Weakness of external rectus on side of lesion. Slow deliberate nystagmus to side of lesion	Same as in unilateral cerebellar tumours	
,, ,, VII.	Paresis slight if present	Paresis more marked	
,, ,, VIII.	Deafness on side of lesion incomplete and variable. Tinnitus general	Deafness on side of lesion marked—generally complete. Tinnitus referred to ear on side of lesion	
,, ,, IX.	Never affected	Occasional paresis on side of lesion	
,, ,, X.	Ditto	Ditto	
,, ,, XI.	Ditto	Ditto	
,, ,, XII.	Ditto	Supranuclear paresis on contralateral side	
Motor system ...	Homolateral paresis, ataxia and atonia	Homolateral paresis and ataxia; contralateral spastic paresis common—occasionally bilateral	Paresis often bilateral, with spasticity. Ataxia general.
Sensory system ...	No change	No change	Occasionally hemianæsthesia.
Reflexes — Tendon	Variable, often diminished	Generally increased, especially on contralateral side	Increased, often unequally.
,, Superficial	Normal	Often diminished on contralateral side	Diminished, often unequally.
,, Plantar	Flexor	Flexor or extensor. Extensor on contralateral or both sides	Extensor on one or both sides.
Sphincters ...	Not affected	Rarely affected ...	Generally affected.

The Relation of the Symptomatology of Cerebellar Disease in Man to the Results of Experiments on Animals.

It is important to consider how far the symptoms of cerebellar tumours are directly due to destructive cerebellar lesions, and to what extent the cerebellar symptoms may be hidden or replaced by irritative or paralytic phenomena according to the extent and degree of pressure the tumour asserts on other parts. This is the more important as many physiologists refuse to accept clinical evidence in discussion of the functions of the cerebellum, owing to the possibility of complication of cerebellar symptoms by indirect or distant lesions.

It seems to us that this is partly unjustifiable. The effect of the increase of intracranial pressure is the first disturbing factor to be considered, but even with infratentorial growths it is fairly uniformly distributed over the whole intracranial cavity and is not liable, except when of long duration, to produce focal disturbance, and then most severely affects the forebrain by dilatation of the ventricles. Its clinical manifestations are well recognised and known to rarely produce such focal symptoms as can be confused with cerebellar disease.

That cerebellar tumours may produce accessory symptoms by direct pressure on the neighbouring structures, especially on the cranial nerves and brain-stem, is evident, but these indirect effects can be easily excluded, and from a knowledge of the symptomatology of lesions of these parts, the true cerebellar signs can be recognised by exclusion. The accumulation of a large amount of clinical observation makes this possible.

Finally, from observation of the effects of destructive lesions entailed by operation (and in several of the cases which succumbed there was destruction of practically the whole of a lateral lobe as complete as is possible by experiment) it was evident that their symptoms were identical in character with those of cerebellar tumour.

It is for various reasons very difficult to compare the

symptoms of cerebellar disease in man with the results obtained by experimentation in the lower animals.

In the first place there is, according to all who have studied the subject, a close relation between the functions of the cerebellum and cerebrum, and the latter is able to compensate, to some degree, for any loss of the former. Thus, while the symptoms of ablation of one cerebellar hemisphere of a dog soon subside under normal conditions, practically no recovery takes place, as Luciani has shown, after extirpation of the contralateral sensorimotor cerebral cortex. The permanent symptoms of a cerebellar lesion must consequently depend in part on the possible extent of this compensation, and as the relative development of the forebrain is greater in man, the power of compensation should, *a priori*, be greater. As illustrative of this may be cited the observations of Luciani and Russell that the symptoms which follow partial lesions of the cerebellum are less intense in the ape than in the dog, and that in the former the compensatory process sets in earlier.

Secondly, the divergence between the results of experimental investigators and the still greater diversity of their interpretations, make it impossible to form a final conclusion on the symptomatology of ablation or stimulation of the various portions of the cerebellum.

Thirdly, the erect attitude of man, the change in the plane of locomotion, and the narrowing of the biped basis, influence the disturbances of locomotion which result from cerebellar disease to such an extent that comparison with the lower mammals is difficult.

And finally, the influence of lesions depends not only on the extent of the destruction they cause, but equally on their rate of onset. In cases of cerebellar tumour of relatively slow growth, there is considerable time and opportunity for compensation by intact parts of the organ (organic) or by other parts of the nervous system (functional). As we, however, possess abundant observations on cases for variable periods after operation, from which large acute lesions resulted, we shall rely mainly on the facts culled from them for comparison with the experimental results.

Luciani, Russell and Thomas record paresis of the

homolateral limbs after ablation of one cerebellar hemisphere, but Ferrier, and Ferrier and Turner were never able to observe it. In several cases, however, there was definite weakness of these limbs when compared with their fellows and it was invariably pronounced after operation, though never of long duration in any great degree. Besides this unilateral paresis, many cases in which the lesions were acute were for some time in an extremely asthenic state, and had little command of muscular power. We are in agreement with Russell that the weakness is as a rule more marked in the upper than in the lower limb, though Luciani describes the leg as the more affected. In several cases of unilateral lesions we have carefully examined the conditions of the spinal muscles, but were rarely able to observe any relative weakness upon either side. In extensive or acute lesions, however, the patient may be unable to hold his head or trunk erect, and this bears out the undue weakness of the trunk muscles which has been observed in animals with extensive cerebellar destruction. As it is possible to exclude affection of the cortico-spinal system as the cause of the weakness, it seems that the available evidence must be accepted as conclusive that homolateral paresis may result directly from cerebellar disease. In man it is easy to make the subject exert his full power and accurately estimate and compare that of the two sides; this is not possible in animals.

In several cases, and especially after operation and in those with acute onset, we found marked hypotonia of the muscles of the homolateral limbs, as Luciani has described. We have, however, never observed any tonic spasm or rigidity in these limbs in the earlier stages, as described by Luciani, Russell and Thomas. On the latter point our observations are in agreement with those of Ferrier, though he did not find hypotonia during any stage after the ablation. The state of the tendon reflexes cannot be regarded as a measure of the tone of the muscles, at least in cerebellar disease. The flaccidity and flail-like character of the limbs when handled, the lack of the normal resistance to passive movement, and the soft and flabby consistence of the muscles to touch, are the indications on which our statements are based.

Astasia, or constant fine tremors with wider oscillation on volitional exertion, is, according to Luciani, the third cardinal symptom of cerebellar lesion, and has also been described by Ferrier and Turner, Russell and Thomas. This has not been a symptom of any of our uncomplicated cases of cerebellar tumour, but in a few cases there was considerable tremor of this variety of the head, trunk, and of the limbs, greater in those of the same side for some days after operation if the parts were unsupported. The unsteadiness only persisted for long in two cases, and was finally limited to the head. An accentuating irregularity of movement of the intention tremor type was then also present, but this was rarely observed in uncomplicated cases of tumour. This condition of tremulousness which follows operation is remarkable and contrasts with the almost unnatural steadiness of the homolateral arm when it is horizontally extended in front of the patient, as described by Babinski, and confirmed by our observations. Reference may here be made to the class of tremor occasionally met with as a clinical symptom, which has been attributed by one of us to lesions of the cerebello-rubral system, though it has not appeared in any of the cases we describe.

Homolateral inco-ordination has been almost universally recognised by experimental investigation as a symptom of cerebellar lesions. Luciani, however, refuses to regard it as an essential symptom of cerebellar disease; according to him, it is due to a combination of the paralytic and compensatory phenomena, while the purposeless and ungraduated actions of the limbs which he styles dysmetria, should result from the asthenia and atonia of the muscles. The facts we have observed do not allow us to agree with these views. True cerebellar ataxy is often demonstrable in chronic cerebellar lesions where there must be a state of equilibrium between the paralytic and compensatory symptoms, and it may be unassociated with asthenia or atonia, though if it is, as we consider it, a primary and essential symptom of destructive lesions of the cerebellum, it will, as a rule, coincide in degree with these.

In man ataxia, atonia and paresis appear to be, and in this order, the essential signs of cerebellar disease.

We have seldom observed in man the pleurotonus, with concavity to the side of the lesion, which most experimenters save Ferrier and Turner have described, though there was often visible, when standing, a forced inclination of the trunk to that side. The position of the head in patients with unilateral cerebellar tumours often corresponds to that in animals after removal of the one cerebellar hemisphere, *i.e.*, the chin is directed to the opposite side, and the head is slightly inclined to the lesion. Neither retraction nor flexion of the head was a marked symptom in any of the cases here considered, but we have observed a pronounced tendency to retraction or falling back of the head when the patient sat up in two cases in which disease of the vermis was diagnosed but not confirmed. As in animals with unilateral cerebellar loss, there is often in man a tendency to abduction of the homolateral leg in walking.

Rotation movements were not a pronounced symptom in any of our cases, but it is necessary to mention the observations of the classical authorities when referring to the subjective and often objective rotation of vertigo and when discussing the significance of the series of rotation fits we describe. Unhappily, so much confusion has arisen from the terminology applied that we find it necessary to translate the descriptions of the forced movements into our own words. By rotation to the right we mean the movement which results if a man while standing erect turns on his vertical axis so that the left shoulder first moves forwards, or, if lying on his back, the left shoulder first rises so that he turns over to his right side.

Besides the confusion from verbal description, there is considerable disagreement among observers on this point. After ablation of one cerebellar lobe Luciani, Ferrier, Turner, Thomas and Muskens would describe the movement in our words as rotation from the side of the lesion, while Russell, Orestano and Pagano, as rotation to the side of the lesion. This forced rotation was only observed for a short time after recovery from the anæsthetic, and was not constant. The

lesion which apparently most frequently results in rotation is simple section of the one middle peduncle, and then, according to Majendie, Ferrier and Curshman, the movement is in the same direction as after ablation of one cerebellar hemisphere, but Schiff and Luciani observed that the animals first rotated towards and later constantly from the side of the lesion, while Hitzig describes the rotation as towards the lesion. More recently Sergi, a pupil of Luciani, has drawn attention to the fact that the direction of rotation is not constant, and may occur in the one or the other direction. As a rule, when one hemisphere is affected, the animal rotates from the side of the lesion, while, if the peduncles are divided, the rotation occurs in the reverse direction. These facts may explain the discrepancies of the various observers, *i.e.*, whether the lesion be of the mass of the cerebellar lobe or of the peduncles, which are largely composed of afferent fibres to the contralateral hemisphere. This is of considerable importance in the consideration of our cases, as in extra-cerebellar tumours the middle peduncle is presumably the part which suffers most.

Russell has found that both section and irritation of the vestibular nerve causes rotation movement in the opposite direction to that which he observed after the removal of one lateral cerebellar lobe.

The most definite rotation-movements we have observed were in the case of cerebellar fits reported, and they were from the side of the lesion towards the healthy side. But in several cases there was a great tendency to lie on or roll over to the healthy side, especially after operation on the one cerebellar hemisphere. These patients would complain of feeling forced to roll over to this side apart entirely from definite subjective vertigo. In states of acute vertigo, on the other hand, where extra-cerebellar tumours compressed the auditory nerve or middle peduncle, the tendency has been to rotate on the vertical axis, as well as to fall towards the side of the disease.

The direction of the subjective rotation in attacks of vertigo, which so frequently occur in both cerebellar and extra-cerebellar tumours, is especially interesting in this

connection. In the former class of case it was always from, in the latter towards, the side of the lesion. This acute vertigo must be regarded as an irritative phenomenon. As the rotation-movements in animals are, from their transient nature and their acute and periodic occurrence, probably of the same nature, our facts are in agreement with the majority of experimentalists excepting Russell as regards intra-cerebellar disease, while our clinical observations agree with his, that lesions of the auditory nerve produce the rotation (in our cases subjective) in the opposite direction to that which occurs in pure cerebellar lesions. It is difficult to understand this inverse relation unless it be due to the fact that each vestibular nerve and middle peduncle has chiefly a crossed connection with the cerebellum.

The apparent movement of external objects has been in both classes of cases from the side of the lesion. This is probably not dependent on a relative displacement of the visual fields secondary to the eye movements, as Mach believes, but is a reflex sensory phenomenon analogous to the sense of rotation of self.

Abnormal positions of the eyes have been observed by most investigators as a more or less persistent symptom after unilateral cerebellar destruction. All are agreed that the eyes are deviated to the opposite side, and in addition, there may be for short periods some irregular position. Luciani and Ferrier describe the typical skew deviation of Majendie, *i.e.*, the homolateral eye is directed downwards and inwards, the contralateral outwards and slightly upwards; but Russell observed the homolateral eye up and out, the other down and out.

We describe the same tendency to conjugate lateral deviation of the eyes to the healthy side in unilateral cerebellar disease, and have in addition repeatedly observed the Majendie "skew-deviation" for a few hours after operation, though in two cases this skew-deviation persisted for longer periods, in one case after operation, in the other apart from operative interference.

Clinical observations agree with Russell's assertion that nystagmus is most marked on looking to the side of the lesion.

It is difficult to compare the biped gait with that of quadrupeds, but as do most observers, we find in man a tendency to fall to the side of the lesion. This does not appear to be entirely due, as Luciani assumes, to paresis of the homolateral limbs, for we have failed to observe any relation between the tendency to fall and such paresis, or that this tendency occurs at the moment the patient throws his weight on the homolateral foot. To us it seems to be rather due to a deficiency in the proper co-operation of the muscles of equilibium, especially in those of the trunk and perhaps in part to paresis of these. The patient does not fall through any giving way of the limb, but as we have frequently observed in acuter cases, the body is definitely inclined towards the affected side till its centre of gravity approaches or exceeds the limit of its base. But the stumbling to the faulty side, as we have observed it, does not depend alone on this factor; there seems to be in addition an inequality in the muscular functions of the two sides, in favour of the contralateral, which results in a disturbance of the equilibrium.

We have not observed in any case of cerebellar disease such sensory loss as has been described by Russell, and one fact deserves special emphasis in view of the assertions of Lewandowsky and others to the contrary, that not even in cases of extensive lesions (*e.g.*, those following operations), was any impairment of the sense of position or of active or passive movements of the limbs to be made out under the most favourable conditions of examination. In man alone can sensibility be accurately tested, and here all evidence conclusively negatives any sensory function of the cerebellum.

The clonic irregularly distributed spasms of the homolateral limbs which we observed or received descriptions of in some cases, are probably analogous to those obtained by Ferrier and Russell (chiefly in the upper extremity), on electrical excitation of one lateral lobe of the cerebellum, and by Pagano on stimulation with curare.

References.

ADLER. *Die Symptomatologie der Kleinhinrkrankungen*, 1899.
FERRIER. "Functions of the Brain," 1876.
FERRIER. BRAIN, 1894.
FERRIER AND TURNER. *Phil. Trans. Royal Society*, vol. 185, 1894.
LUCIANI. *Il Cervelletto*, 1892.
LUCIANI. *Ergebnisse der Physiologie*, Dritter Jahrgang, 1904, abth. ii.
MUSKENS. *Journal of Physiology*, vol. xxxi.
ORESTANO. *Rivista di pathologia nervosa e mentale*, 1901.
PAGANO. *Rivista di pathologia nervosa e mentale*, 1902.
RISIEN RUSSELL. *Phil. Trans. Royal Society*, vol. 185, 1894.
RISIEN RUSSELL. *British Medical Journal*, 1895.
RISIEN RUSSELL. *British Medical Journal*, 1897.
SERGI. *Rivista sperment. di Freniatria*, xxix.

Description of Plates.

Figures 1, 2 and 3 are photographs of the brain of case 20 to represent the position of extra-cerebellar tumours and their relations to the neighbouring parts.

The sections represented in figs. 2 and 3 are not quite vertical to the plane of the brain-stem, but oblique from above, downwards and forwards consequently the degree of compression of the pons relative to that of the cerebellum appears greater than is actually the case.

Fig. 1 shows the position of the tumour on the base of the brain and the compression of the pons, and of the right lateral lobe of the cerebellum by it.

Fig. 2 is a section through the cerebellum and medulla oblongata at the level B, B, fig. 1. Seen from in front. It illustrates the compression of the right lateral lobe of the cerebellum by the tumour.

Fig. 3 is a section through the brain at the level represented by the line A, A in fig. 1. Seen from behind. It illustrates the compression of the middle cerebellar peduncle, and of the right lateral lobe of the cerebellum, and the backward displacement of the latter.

Fig. 4 is a photograph of case 7 taken a few weeks after removal of a tumour from the left lateral lobe of the cerebellum, to illustrate skew-deviation of the eyes.

PLATE I.

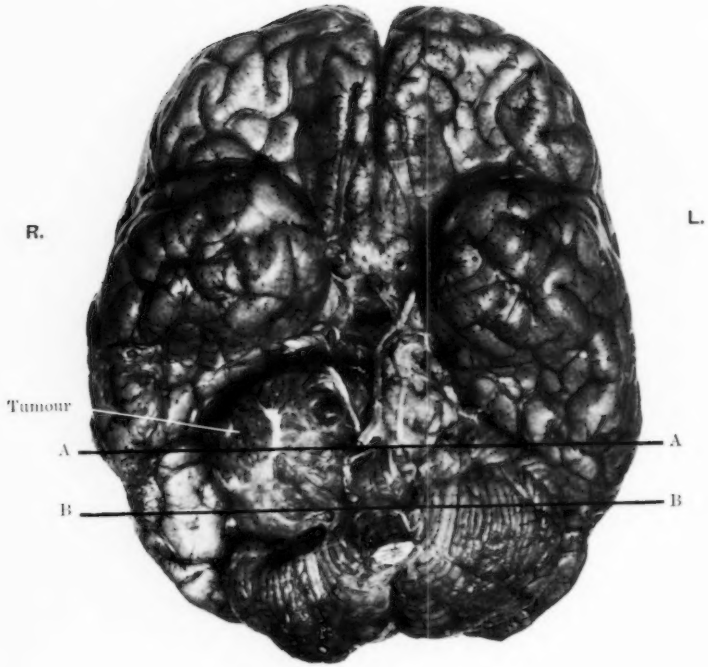

FIG. 1.

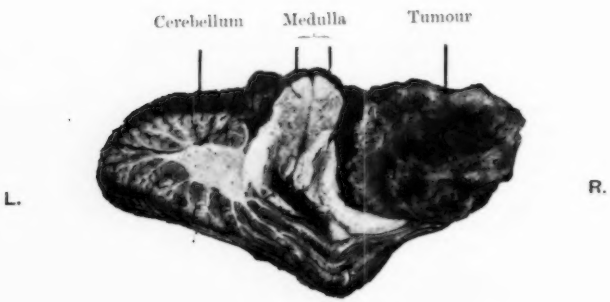

FIG. 2.

PLATE II.

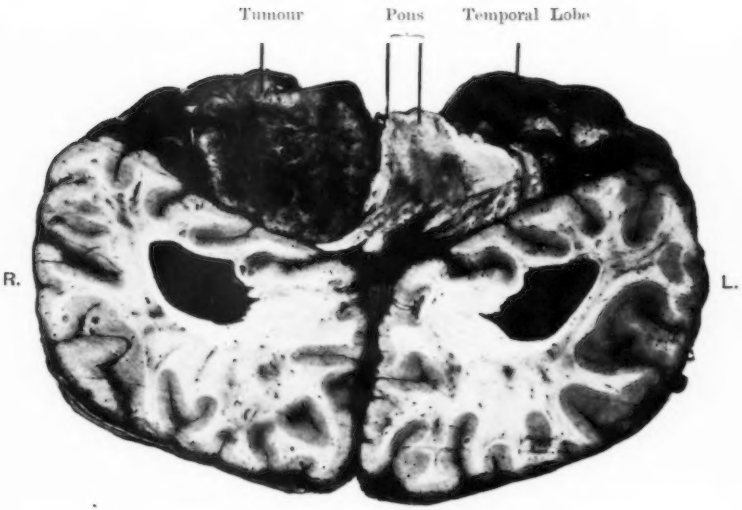

Fig. 3.

Fig. 4.

Appendix.

Reports of 22 Cases of Cerebellar and Extra-cerebellar Tumour which have been personally observed, and in which the Diagnosis has been confirmed by Operation or by Autopsy.

Case 1.—*Tubercular tumour of the right lateral lobe of the cerebellum.*

R. B., aged 24, female. Under the care of Sir William Gowers, 1904.

Family history and previous health unimportant.

For seven months headache, chiefly in the occipital region, and from there pain often radiated down her neck; frequently accompanied by attacks of vomiting. For five months had walked like a drunken person, staggered and fell, generally to the R. For four months her hands had been weak and unsteady, and she had held her head stiffly over to the R., and complained of diplopia, especially on looking to the R.

Had several fainting attacks in which she became cyanosed and fell backwards, generally associated with vomiting but never accompanied by convulsions.

Special senses.—Smell and taste normal. Hearing equally good on both sides, never tinnitus or vertigo. Vision very poor. Intense optic neuritis passing into atrophy.

C. N., 3, 4, 6.—Constant slight deviation of both eyes to the L. Slight weakness of R. external rectus.

Nystagmus on lateral movement, slow and forcible on looking to R., finer and more rapid on looking to L. It was not constantly present.

Pupils equal, reactions good.

7.—Slight weakness of the R. side of face on both expressional and volitional movement.

5, 9, 10, 11, 12.—Functions, normal.

Attitude.—Head generally retracted with occiput approximated to the R. shoulder. No other peculiarity.

M. S.—Muscles soft and flabby, especially those of the R. limbs which were definitely hypotonic. Strength of both limbs poor, R. considerably weaker than L. Co-ordination of finer movements

impaired on both sides, considerably more on the R. than on the L. Pronation-supination test poorly executed with R. arm. No tremor or spontaneous movements.

Gait.—The R. shoulder was generally held higher than, and in front of, the L. No lordosis. The R. leg was abducted and rotated outward so that she stood on an unnaturally broad base. She could stand better on the L. leg than on the R. She tended to deviate to the R. of the line of progression and stumbled and fell to the same side. Less frequently the L. shoulder was held in front of the R., and she staggered and stumbled to the L.

Sensory system.—No defect in any form of sensation.

Reflexes.—Deep: Variable, the L. however being constantly brisker than the R., which were occasionally absent. Superficial reflexes equally brisk. Plantars flexor.

Cranium.—No bulging or local tenderness. No bruit audible on auscultation.

Ten days after admission the tumour was removed by Sir Victor Horsley. It was an almost spherical mass measuring about $3\frac{1}{2}$ cms. in diameter, and lay in the R. lateral lobe of the cerebellum. The patient recovered rapidly from the operation.

For some days after, the phenomena above described were exaggerated, especially the weakness, hypotonia and ataxy of the R. limbs, and the tendon-reflexes were absent. From this time the patient gradually improved and there was considerable return of vision. After some weeks she was able to walk fairly well, though the gait was still reeling with a constant tendency to deviate and fall to the R. The constant conjugate deviation of the eyes to the L. was more marked and there was very slow, coarse nystagmus on lateral deviation to the R. The superficial reflexes remained normal; the deep were for a time absent on the R. and diminished on the L., but afterwards became brisk, though always more so on the L. than on the R.

Two months after the operation the patient developed tubercular meningitis and died. At the autopsy it was found that the greater part of the R. lateral lobe of the cerebellum had been destroyed.

Case 2.—*Tumour* (*glioma*) *of the left lateral lobe of the cerebellum.*

A. N., aged 27, female. Under the care of Dr. Ferrier. 1904.

The patient's family history was good, but she herself suffered from pulmonary tuberculosis and from disease of the R. middle ear. Three months before admission to the hospital she had

severe pain in the back of the head and neck, associated with attacks of vomiting and "rushing noises" in the back of the head. Shortly after the onset of these symptoms diplopia developed. A month later her eyesight began to fail, and her hearing became more impaired. Three weeks before admission her walking became very unsteady, and she had attacks of giddiness, in which external objects seemed to move from L. to R. in front of her.

State on Admission.

A pale, delicate woman, with tubercular affection of both apices pulmonum.

Special senses.—Smell and taste normal. Hearing.—Bone conduction was much better on the R. side than on the L., though, owing to the condition of the middle ear on the R. side, aerial conduction was better on the L. than on the R. Vision.—R., $\frac{6}{18}$; L., $\frac{6}{18}$. Intense optic neuritis.

C. N., 3, 4, 6.—Weakness of L. external rectus. Slow and deliberate nystagmus on lateral movement in both directions, more marked to L. On downward movement the nystagmus was vertical, while on upward movement it was vertical, with a rotation to the L. Pupils were dilated, equal and reacted normally.

7.—Slight weakness of the L. side of the face.

5, 9, 10, 11, 12.—Functions normal.

Attitude.—Showed no peculiarity.

M. S.—Muscles were of equal size and natural in tone. The strength of the limbs was equal on the two sides. Co-ordination was definitely impaired in the L. limbs. Babinski's cerebellar sign was not definite. There was no tremor when at rest.

Gait.—She deviated to the R. but staggered to the L. She could stand better on the R. than on the L., and when her feet were placed together she tended to fall backwards and to the L.

Sensory S.—No defect.

Reflexes.—Deep; were brisk and equal on both sides. The superficial reflexes were normal. The plantar reflexes were flexor, and there was no sphincter affection.

Cranium.—There was no deformity or bulging, but the occipital region was tender on pressure, especially on the L. side. Mr. Ballance operated, and removed a large tumour from the left lateral lobe of the cerebellum in its posterio-inferior angle, just above the fourth ventricle.

The patient collapsed and died the same evening.

Case 3.—*Tumour* (*tubercular*) *of the left lateral lobe of the cerebellum.*

H. B., aged 18, male. Under the care of Dr. Buzzard. 1904.

This patient's mother had had syphilis, and he had tubercular disease of the L. hip and the R. wrist.

A year ago patient suffered from several severe attacks of occipital pain, which radiated down his neck. This, however, gradually passed off, and he felt well till five weeks ago, when the headache returned, accompanied by vomiting and vertigo. At these times he felt as if he were being twisted round L. to R., and external objects appeared to move in the same direction. He also had double vision on looking to the L., and noticed that he was becoming deaf in the L. ear. About a week later his walking became affected, and he staggered and tended to run into people on his L. side. On several occasions he complained of twitching of the L. side of his face.

State on Admission.

There was latent tubercular disease of both the R. wrist and L. hip, and the corresponding muscles were much wasted.

Special senses.—Smell and taste normal. Hearing.—Slight deafness in L. ear. Vision.—R., $\frac{6}{12}$; L., $\frac{6}{9}$. Well marked optic neuritis.

C. N., 3, 4, 6.—Weakness of both external recti, greater of the L. Nystagmus, slow, coarse and deliberate on looking to the L., fine and rapid on looking to the R. Vertical irregular nystagmus was also present on upward movement. The pupils were equal and reacted well.

5.—Slight wasting of the L. masseter, with deviation of jaw to L.

7, 9, 10, 12.—Functions normal.

Attitude.—The occiput was constantly approximated to the R. shoulder, the chin pointing to the L.

M. S.—Owing to the joint conditions the relative strength of the muscles could not be estimated. Tone of L. limbs subnormal. Co-ordination impaired in both upper extremities, and slightly in the lower, the ataxy being greater in the L. limbs. Babinski's cerebellar sign was not definite. Hands held very steady, especially L., when horizontally extended in front of him.

Gait.—Was reeling and unsteady, and he deviated and stumbled more to the L. than to the R.

Reflexes.—Deep; all diminished on the R., normal on the L.

Superficial; were brisk and equal; plantars flexor, and there was no sphincter trouble.

Sensory S.—Unaffected.

Cranium.—No bulging or tenderness.

The patient remained in much the same condition while under observation, except for an increase of the optic neuritis.

He was operated on by Sir Victor Horsley, the second stage being completed on May 3, 1904, when a tubercular tumour of irregular shape, which measured about four cms. in all diameters, was removed from the L. lateral lobe of the cerebellum. It came to the surface on the dorsal aspect. The patient stood the operation well.

May 6, 1904.—Symptoms and signs as before operation, except for definite weakness of the L. side.

May 22, 1904.—All signs less marked, but still present. The gait nearly natural. The deep reflexes brisk and equal, and the plantar responses flexor.

Six weeks after operation most of the previous symptoms were still present, but there was in addition weakness of the lateral deviation of the eyes to the L., and slow, forcible nystagmus on attempting it. The facial and masseter weakness had almost disappeared. There was slightly greater weakness, ataxy and inco-ordination of the L. limbs. Gait as before operation. Tendon reflexes diminished on L. Gait became eventually normal, except for the complications due to the tubercular affection of the L. hip.

Case 4.—Cyst in left lateral lobe of the cerebellum.

S. C., aged 7, male. Under the care of Dr. Ormerod. 1903.

Patient's previous health and family history were good.

Three weeks previous to his admission he began to suffer from headache and vomiting, and a few days later complained of dimness of sight. His condition rapidly became worse, and he developed a squint with diplopia, and staggered about when he walked. He also complained of transient attacks of blindness. The headache was chiefly in the L. frontal and L. occipital regions.

State on Admission.

Patient very dull and drowsy. Viscera healthy.

Special senses.—Smell and taste normal; hearing, diminished on L. Vision, only light perception. Intense optic neuritis.

C. N., 3, 4, 6.—Eyes deviated to the R.; weakness of the L.

external rectus, other ocular movements good. Slow, deliberate and coarse nystagmus on looking to L., fine and rapid on looking to R.

7.—Slight weakness of L. lower face.

12.—Tongue protruded slightly to the L.

5, 9, 10, 11.—Functions normal.

Attitude.—In bed he lay on his L. side, with the occiput approximated to the L. shoulder.

M. S.—The muscles of good size. The L. limbs much weaker and more flaccid than the R. Co-ordination much impaired in both L. limbs and to a less extent in the R. upper extremity.

Gait.—There was well-marked lordosis when he stood. He walked upon a wide base, the L. leg being somewhat abducted and rotated outward. He tended to drag the R. foot. Very ataxic, staggered to either side, and tended to fall to the right.

Sensory S.—Unaffected.

Reflexes.—Deep; brisk both sides. Superficial; abdominal diminished on the R., brisk on the L. Plantars flexor.

Cranium.—Slight bulging in L. occipital region, with tenderness on pressure.

A large cyst in the L. lateral lobe was opened and drained by Sir Victor Horsley. A very rapid recovery took place, and patient could walk a fortnight later. A month after operation his vision was $\frac{6}{6}$ in both eyes; the discs were muddy, but not swollen. There was slight lordosis and ataxy of the L. side. Slow nystagmus on looking to the L. The power of the L. side was much improved.

Six months after operation the only abnormal sign was a slight degree of lordosis. Reflexes normal.

Case 5.—Cyst of the left lateral lobe of the cerebellum.

A. W., aged 15, male. Under the care of Dr. Tooth. 1903.

The previous history unimportant.

Four months before admission patient suffered constantly from severe headache in the occipital region and had frequent attacks of vomiting. These symptoms were soon followed by dimness of vision and periods of transient blindness. His walking became affected a few days later, he staggered like a drunken man. Three months later he began to suffer from attacks of giddiness, in which he had no sense of rotation either of himself or his surroundings, but merely a feeling as if he must fall. During the last month he noticed unsteadiness of the L. arm, and in a less degree of the R. arm.

State on Admission.

Special senses.—Smell, taste, and hearing normal. Vision, R., $\frac{6}{36}$; L., $\frac{6}{18}$. Intense optic neuritis.

C. N., 3, 4, 6.—Slight weakness of L. external rectus. Ocular movements good. Nystagmus; coarse and deliberate on lateral movement to the L., more rapid on looking to the R. Pupils large, equal, reacted well.

5, 7, 9, 10, 12.—Functions normal.

Attitude.—Head generally rotated to the R., with the occiput approximated to the L. shoulder.

M. S.—Muscles were well developed and neither rigid nor flaccid. The strength of the L. limbs was considerably less than that of the R. Co-ordination was much impaired in the L. limbs, and slightly affected in the R. arm. Babinski's cerebellar sign was present in the L. arm. There was no tremor when at rest.

Gait.—The body was held slightly rotated to the R., the L. shoulder being in front of the R. There was slight lordosis. He walked in a staggering and drunken manner, but did not deviate to either side.

Sensory S.—Normal.

Reflexes.—Deep; all brisk and equal. Superficial; normal. Plantars flexor. No sphincter trouble.

Cranium.—No bulging, deformity, or tenderness.

Sir Victor Horsley removed a large cyst which lay deeply situated in the L. lateral lobe. It did not appear to be connected with any growth. The patient recovered well from the operation.

Headache and vomiting at once ceased, and other symptoms were relieved. For some days there was some weakness of conjugate movement of eyes to L., and very marked nystagmus on movement, especially to L.; also spontaneous nystagmus, with jerking to L. There was considerable paresis, atonia and ataxia of the L. limbs, but these symptoms soon disappeared. The L. tendon-reflexes were for a time diminished. Three weeks after operation he was able to walk alone, though in a staggering manner, and with a great tendency to stumble and deviate to the L.

Six months after operation all symptoms had disappeared, except some lordosis and a tendency to occasionally stumble to L. Later, his gait was quite normal.

Case 6.—*Cyst in the left lateral lobe of the cerebellum.*

E. B., aged 14, female. Under the care of Dr. Ferrier, 1904. The patient's previous health and family history, good.

The present illness came on three and a half months ago with headache and vomiting. The headache was bilateral and both occipital and frontal in situation; at first occasional, but latterly almost constant. Also giddiness, external objects appearing to go round in front of her though not in any particular direction. Her vision became weak about a month ago and rapidly deteriorated, and about the same time it was noticed her head was held stiff and was bent over towards the L. Diplopia also developed about this time. Fourteen days ago her gait became affected, she staggered and tended to walk to the L. For the last seven days she has complained of noises in both ears.

State on Admission.

Special senses.—Smell, taste and hearing, normal. Vision.—R., $\frac{6}{18}$, L., only light perception. Intense optic neuritis.

C. N., 3, 4, 6.—Ocular movements good, except slight weakness of L. external rectus; occasionally coarse nystagmus on looking to L. Pupils equal and reacted well.

7.—Slight weakness of the L. side of the face.

5, 9, 10, 11, 12.—Functions normal.

Attitude.—Occiput approximated to L. shoulder and chin pointed to R. Lay on R. side. Lumbar lordosis when erect.

M. S.—Development and tone of muscles, good. Considerable weakness of L. limbs, which were also ataxic, though the co-ordination of R. was good. Executed rapidly repeated movements equally well with both arms. No tremor or unsteadiness of hands.

Gait.—Rotation of vertebral column to R., *i.e.*, L. shoulder held in front of R. Could walk fairly well, but deviated and stumbled to L. Stood more steadily on R. foot than on L.

S. S.—Unaffected.

Reflexes.—Deep: Arm-jerks diminished and equal. Knee-jerks—R. diminished, L. absent. Both ankle-jerks diminished. The superficial reflexes brisk and equal and the plantars flexor. No sphincter affection.

Cranium.—Slight prominence in the L. occipital region, with great tenderness on pressure.

A few days after admission Sir Victor Horsley evacuated a cyst in the L. lateral lobe, which contained some clear straw-coloured fluid. The patient bore the operation well. A fortnight later the optic neuritis was rapidly subsiding with improvement of vision. The eyes were constantly deviated to the R. Weakness of conjugate deviation to L. Slow, deliberate nystagmus on

looking to L. Head in position described above; limbs flaccid, especially the L., and the weakness and ataxy of the L. limbs more marked. The deep reflexes present on the R. absent on the L. Plantar reflexes normal. Patient unable to walk alone. A month after this paresis and ataxy of L. limbs was less. She could walk alone, but deviated and stumbled to L. Considerable lordosis in erect attitude. Ocular movements normal.

Six months after operation patient was able to walk quite well, but still tended to stumble to L. Reflexes, normal. The paresis and ataxia of L. limbs had disappeared.

Case 7.—Gumma of the left lateral lobe of the cerebellum.

H. M., aged 27, male. Under the care of Dr. Buzzard, 1904. Patient had previously malarial fever and syphilis.

For two years attacks of occipital headache accompanied by vomiting and frequently by vertigo, in which he felt as if he himself and the external objects moved from L. to R. Also, noises in his L. ear. A few months later his gait became affected, he staggered and stumbled to the L. It was also noticed that when he stood up there was marked lordosis. Two months before admission all his symptoms became more intense, he had diplopia and failure of vision and occasional attacks of transient blindness. For a month he had lain in a comatose state.

State on Admission.

He was very drowsy and his respiration was periodic.

Special senses.—Smell and taste normal. Hearing, slight deafness on L. side. Vision much impaired; intense optic neuritis.

C. N., 3, 4, 6.—Weakness of the L. external rectus. Slow, deliberate jerking nystagmus on looking to L. Pupils large, equal reacted slowly.

5.—Slight weakness of the L. masseter and also slight L. hypoæsthesia.

7.—Weakness of the R. lower face.

9, 10, 11, 12.—Articulation slow and swallowing difficult, but no paresis of palate or vocal cords.

Attitude.—In bed he lay rather on his L. side with the occiput approximated to the L. shoulder. When he sat up this position of head was still maintained, and he complained of vertigo, subjective rotation of self and objects from L. to R.

M. S.—Owing to syphilitic changes in both elbow-joints he could not extend his fore-arms fully. There was no muscular

wasting apart from that due to these changes. The muscles of the R. side were normal in tone, but on the L. definitely hypotonic. The L. limbs were slightly weaker than the R. Co-ordination was slightly impaired in the R. arm and markedly so in the L. limbs. Babinski's cerebellar sign was present in L. There was no spontaneous tremor.

Gait.—Owing to patient's condition it was judged to be inadvisable to get him out of bed.

Sensory system.— No defect.

Reflexes.—Deep; arm-jerks were present on both sides, L. diminished; knee and ankle-jerks were diminished on the R., absent on the L. The superficial reflexes were brisk and equal on two sides, except L. corneal reflex, which was impaired. Plantar responses, flexor. No sphincter trouble.

Cranium.—No deformity, but tenderness on pressure over L. cerebellar region.

Sir Victor Horsley opened the skull in the L. cerebellar region the following day, and three days later he completed the operation. A gummatous mass which occupied the L. lateral lobe especially in its dorsal and outer aspects, where it had infiltrated the dura, was removed.

Immediately after the operation there was skew-deviation of eyes, the L. being directed downwards and inwards, the R. outwards and slightly upwards (plate ii., fig. 4). Slow nystagmus of both eyes in all movements, also spontaneous nystagmus to L.

Tendon-reflexes diminished on R. side, absent L. When examined next day the position of the eyes was unaltered. Upward and outward movement of L. eye limited, inward of R. impaired. Fine, rapid nystagmus on looking to R., while to the L. the nystagmus was more forcible, slow and deliberate. Pupils dilated. Paresis of L. masseter and face. Impairment of hearing on L. Muscle tone of R. limbs subnormal, L. limbs very hypotonic and flail-like when handled. Great weakness and ataxy of L. limb. Tendon-reflexes diminished on R., absent on L. No sensory change.

A month after operation the skew-deviation of the eyes still persisted; nystagmus unaltered. Weakness of L. trigeminus had disappeared and that of L. external rectus and face was less marked. Optic neuritis subsiding and sight improving. Still considerable paresis, hypotonicity and ataxy of L. limbs, while the R. were almost normal. Unable to stand. Tendon reflexes diminished in R. limbs, absent in L. Plantar flexor responses. Occasionally forced rotation to R. Two months after operation he

was much stronger, no headache, but frequently vomited. On pressing on wound vertigo, the subjective rotation of external objects and self being from L. to R. Occasionally forced rotation to R. Vision improved to $\frac{6}{9}$. Optic neuritis completely subsided.

Skew-deviation of eyes had almost disappeared, but still weakness of conjugate movement of eyes to L. Nystagmus slow and coarse to L., fine and rapid to R.

No peculiarity in attitude except slight lordosis and tendency to lean to L. Paresis, hypotonicity and ataxia of L. limbs much less. Could not execute rapidly repeated movements with L. arm. Could just walk alone, inclined trunk to L., and deviated, stumbled and fell to L. Rotation of trunk to R., *i.e.*, L. shoulder forwards.

Tendon-reflexes brisk, L. slightly greater than R. Intermittent, irregular ankle clonus on L. Skin reflexes, normal.

Case 8.—*Tumour* (*glioma*) *involving the middle lobe and in part the lateral lobes of the cerebellum.*

E. B., aged 14, female. Under the care of Dr. Bastian. 1904.

Patient's family history and previous health unimportant.

For eighteen months she had severe pain in the occipital and R. temporal regions, associated with vomiting, retching and nausea. These symptoms were very constant and severe. Eight months ago her eye-sight began to fail, and she suffered a good deal from noises in her head "like bells ringing," but no deafness. Four months later her hands became unsteady, especially the R., and two months later her walking was affected. She staggered like a drunken person, more often to the R. than to the L.

During the last ten months she frequently had attacks of vertigo, generally a feeling as if she and the room were revolving from L. to R., although occasionally it was in the reverse direction.

State on Admission.

Special senses.—Smell and taste normal. Hearing.—Slight deafness L. ear. Vision.—R., $\frac{6}{9}$; L., $\frac{6}{12}$. Intense optic neuritis.

C. N., 3, 4, 6.—Weakness of deviation of eyes to L. Occasionally nystagmus on lateral movement of eyes, especially to L. Pupils equal, reacted normally.

5.—Slight loss of sensation L.

7.—Weakness of L. face, both expressional and volitional.

9, 10, 11, 12.—Functions normal.

Attitude.—Occiput approximated to L. shoulder; R. shoulder held higher and in front of L.

M. S.—No rigidity or flaccidity of limbs. Strength poor on both sides, especially of L. limbs. Inco-ordination of both sides, greater L. than R. Difficulty in rapidly repeating movements with arms, especially L.

Gait.—Stood with body bent over to the L. and feet apart; staggered, reeled and fell to both sides, more frequently to L.

She stands better on the R. leg than on the L.

Sensory S.—Unaffected, save on face.

Reflexes.—Deep; arm jerks equal and brisk, knee and ankle jerks absent. Superficial; present more R. than L. Plantars flexor. No sphincter affection.

Cranium.—No bulging. Tenderness on pressure over occipital region.

During the next month the deep reflexes were at times brisk and equal, but again disappeared, and there was a definite increase in the weakness and inco-ordination of the L. side. The optic neuritis had also increased.

Sir Victor Horsley then removed, in small pieces, a large tumour, which lay in the medial portion of the left lateral lobe, in the inferior portion of the vermis, and extended into the R. lateral lobe at a deeper level than on the L.

The patient recovered well from the operation, but for many days after there was a condition of general asthenia and loss of all deep reflexes, also extreme inco-ordination of all limbs, and inability to sit up or stand.

During the next month the patient's condition improved; there was a steady return of strength, and co-ordination and the deep reflexes reappeared. Two months after the operation the weakness and ataxia were almost limited to the L. side. Slow and deliberate nystagmus on looking to the L., fine and rapid to the R.

The optic neuritis had subsided. The other cranial nerve functions were normal. She could walk with assistance, but tended to stagger and fall to the L., sometimes backwards and to the L.

After five months there was little further improvement. Occasionally headache and frequently vertigo on abrupt movement, the subjective movement of objects and of self being from L. to R., and often forced movements, rolling over to R. side. The nystagmus persisted; L. limbs very flaccid and weaker than R. Power poorly sustained. Ataxy of L. limbs, and some intention-

tremor of R. Could only walk with assistance, deviated to L., and fell to either side, most frequently to L. Tendon reflexes brisk. Mental state fair.

Case 9.—*Tumour (glioma) of the vermis cerebelli.*

C. W., aged 6 years, male. Under the care of Dr. Risien Russell. 1902.

The patient had never been strong and had suffered from rickets. The present illness came on abruptly three weeks before his admission to hospital. He suffered from frontal headache, vomiting, and unsteadiness in walking, his sight became misty, and his mother noticed that his eyes were "queer" and that his hearing was impaired. His condition progressed rapidly and he became drowsy and irritable and frequently soiled himself.

State on Admission.

Very drowsy and sleepy. Severe headache.

Special senses.—Smell and taste, normal. Hearing, impaired on the L.

Vision.—Could only count fingers at ten feet. Severe optic neuritis in both eyes.

C. N., 3, 4, 6.—Weakness of both external recti. Coarse, slow, deliberate nystagmus on lateral deviation to either side, more so to L. than to R. Pupils large and equal, reacted normally to light and convergence.

5, 7, 9, 10, 11, 12.—Functions normal.

Attitude.—When in bed he always lay on his R. side with his head somewhat retracted. When standing there was no peculiarity in the way he held his head.

M. S.—Muscles were well developed, tone was deficient. The strength on both sides was fair and about equal. Co-ordination was impaired on both sides, more so on the L. There was no tremor when at rest.

Gait.—He stood with considerable lordosis, and his feet wide apart. He reeled and staggered to both sides, but more frequently to the L. When his feet were placed together he tended to fall backwards and to the L.

Sensory S.—No defect was noted.

Reflexes.—Deep: increased on both sides, especially on the L. Superficial reflexes present on both sides. The plantar reflexes extensor. No sphincter trouble.

Cranium.—Definite bulging in the L. occipital region with tenderness on pressure. Percussion of the skull yielded a "cracked-pot" note. Considerable hydrocephalus.

Two days after admission the patient became unconscious, his breathing periodic, and the pulse small and rapid. All the reflexes were absent. This condition passed off in about two hours time. On the following day Mr. Ballance opened the skull over the L. cerebellar region. No evidence of tumour in the L. lobe was found, although before the dura was opened there was great intradural tension and a large quantity of cerebro-spinal fluid escaped when it was incised. The after history of this case is of interest, as the patient had a series of fits which were carefully observed. The patient died the day after the operation, and at the autopsy it was seen that there was considerable destruction of the L. lateral lobe of the cerebellum and a hæmorrhage in the region of the nucleus dentatus. A large tumour measuring about $3\frac{1}{2}$ cms. across lay in the central portion of the vermis, extending between the middle cerebellar peduncles, but further to the L. than to the R.

Case 10.—*Tumour* (*glioma*) *involving the vermis and both lateral lobes of the cerebellum, chiefly right.*

M. McC., aged 8, female. Under the care of Dr. Risien Russell. 1904.

The family history and previous health of the patient unimportant.

For fifteen months had suffered from constant severe headache and vomiting, the pain often radiating into her neck. This lasted for a month, but recurred again two months later. Shortly after this her eyesight began to fail and her walking became affected; she staggered about like a drunken person. For eight months before admission she was unable to walk, and noticed that the R. hand was unsteady. She never had vertigo or auditory disturbances.

State on Admission.

Special senses.—Smell, taste, and hearing normal. Vision.—R., blind; L., could count fingers. Post-neuritic atrophy of optic discs.

C. N., 3, 4, 6.—Weakness of R. external rectus, and slighter of L. Slow, jerky nystagmus on lateral deviation to the R. Pupils large, the R. inactive to light; the L. reacted sluggishly.

5, 7, 9, 10, 12.—Functions normal.

Attitude.—The occiput held approximated to the R. shoulder, and the chin directed to the L.

M. S.—Muscles were large, and the tone and strength were good on the two sides. Co-ordination was impaired in both R. limbs, and to a lesser degree in the L. upper extremity.

There was no tremor in the extended and unsupported limbs.

Gait.—There was considerable lordosis when erect. The R. leg was abducted and rotated outwards, and she used it awkwardly. The R. shoulder was generally kept in front of the L. She could only walk with help, and tended to fall backwards and to the R. She was able to run fairly well.

Sensory S.—Unaffected.

Reflexes.—Deep; all increased on both sides. The superficial reflexes diminished on both sides. The plantar reflexes extensor. There was no sphincter trouble.

Cranium.—Considerable hydrocephalus, with bulging of the R. occipital region, and tenderness on pressure over it.

Sir Victor Horsley operated three days after admission. The skull was thinned in the R. occipital region, and a large tumour was found in the R. lateral lobe, and extending into the middle lobe. The greater part of this was removed.

The patient stood the operation well. There was later weakness of deviation of the eyes to the R. with nystagmus. The limbs, especially the R., were very hypotonic and weaker and more ataxic than before operation. The deep reflexes were diminished on the R. Recovery seemed well assured till three weeks later, when she suddenly died.

At the autopsy it was found that only part of the tumour had been removed. A large, spherical mass of soft consistence lay in, and practically destroyed the greater part of the middle lobe, and extended into both lateral lobes, chiefly into the posterior part of the R.

Case 11.—*Tumour* (*cystic glioma*) *of the middle lobe of the cerebellum.*

W. W., aged 26, male. Under the care of Sir William Gowers. 1902.

The family history and previous health of the patient unimportant. Three years previous to his admission to hospital, he had suffered from severe headache and vomiting. He was admitted to a general hospital, and trephined over the L. cerebellar region for the relief of those symptoms. This gave him freedom from the headache for some months, but owing to its recurrence he was again trephined a year later, this time over the R. cerebellar region, but without relief. Two months later the L. lateral

lobe of the cerebellum was opened, and a cystic tumour found and removed. After nine months, as the headache had returned, the L. cerebellar region was again explored and some more tissue removed, with, however, only temporary relief. Since then and up to the time of his admission into the National Hospital he had suffered from L. occipital headache, vomiting, staggering gait and attacks of giddiness.

On admission.—Smell, taste, and hearing normal. Vision poor. There was in the eyes a fresh neuritis on the top of a secondary atrophy.

C. N., 3, 4, 6.—No ocular palsy, no nystagmus. Pupils equal and reacted well.

5, 7.—There was slight impairment of the motor functions of these nerves on the R. side.

Attitude.—Owing to constant severe headache when he lay flat, the patient always had his head raised when in bed, and usually lay on his R. side. When standing, the occiput was approximated to his R. shoulder.

M. S.—The tone and development of the muscles were good on both sides. The strength of the two sides was about equal. Co-ordination was impaired on both sides, more on the R. than the L. There was no tremor when the limbs were horizontally extended in front of him.

Gait.—Was reeling and staggering to both sides. Frequently vomiting on movement.

Sensory S.—Unaffected.

Reflexes.—Deep; all increased, R. more than L. Superficial reflexes were normal. Plantars flexor. No sphincter affection.

Cranium.—In L. occipital region there was a trephine opening, the size of a crown-piece, through which a soft pulsating tumour protruded. On the R. side there was a smaller trephine opening, but no bulging. Sir Victor Horsley operated some weeks later, and exposed the dura mater more freely over both sides of the cerebellum, but no tumour was seen. There was considerable hæmorrhage and shock; the dura was incised, and the wound closed. The patient was much collapsed, and died several hours later. The autopsy revealed a large cystic growth, which involved the greater part of the middle lobe and the median portion of the dorsal surface of each lateral lobe, especially of the R.

Case 12.—*Tumour* (*glioma*), *left extra-cerebellar.*

S. R., aged 42 years, female. Under the care of Dr. Beevor. 1903.

Previous health and family history good.

Six months before admission to hospital she began to reel about as if drunk when walking, and her gait rapidly became worse. She also had severe headache, generally occipital, associated with pain and stiffness in neck, and often vomiting. Frequent attacks of giddiness, in which external objects seemed to move in front of her from L. to R., while there was a sense of rotation of self on her vertical axis in the opposite direction. If standing would fall at once, generally forward and to the L. Later the character of the vertigo changed; everything would seem to grow dark, severe tinnitus in L. ear and violent headache set in; then she would vomit; during these attacks would feel either very cold and shiver, or flushed and hot, and sweated profusely. For six months she had complained of deafness and tinnitus in the L. ear. Recently had transient amblyopia.

State on Admission.

Special senses.—Smell and taste normal. Hearing, R. good; L. lost. Sight, could only count fingers at a short distance. Intense optic neuritis.

C. N., 3, 4, 6.—No diplopia or squint, but full lateral deviation of the eyes to the L. was difficult; they could not be long maintained in that position, and quickly receded medialwards. Nystagmus on lateral movements, slower and coarser on looking to the L. than to R.

7.—Slight weakness of L. side of face of peripheral type.

5, 9, 10, 11, 12.—Normal.

Attitude.—R. ear approximated to R. shoulder, but this position not constant. Considerable lumbar lordosis when in the erect position.

M. S.—Muscles well developed, tone and strength good. Well-marked inco-ordination of the L. limbs, chiefly of the arm, and slight unsteadiness of the R. hand in finer movements.

Gait.—Very reeling and unsteady of the drunken type, considerable lordosis; L. shoulder held in front of and higher than R. Constantly deviated and stumbled to the L. Base rather wide. Stands much better on the R. foot alone than on the L.

Sensory S.—No change in any form of sensation detected.

Reflexes.—All normal, and equal on the two sides. Plantars flexor.

Cranium.—No deformity. Some tenderness on pressure in the occipital region.

During the next ten days the patient became much weaker,

and on a few occasions fell into a state of coma after severe attacks of headache. Sir Victor Horsley attempted to remove the tumour, but the patient died in a sudden attack of coma four days after the first stage of the operation, *i.e.*, opening of the skull.

At the autopsy a small firm ovoid tumour about three cms. in diameter was found, attached by a pedicle to the membranes under the L. lateral lobe of the cerebellum.

Case 13.—*Tumour* (*fibroma*), *left extra-cerebellar.*

C. H., aged 56 years, female. Sir William Gowers. 1903.

Had good health previous to present illness.

For two years had occipital headache and pain in back of neck, which increased in frequency and severity. Occasional attacks of vertigo, in which she tended to rotate and fall to L.

Also deafness of L. ear with tinnitus. During the three months previous to admission there was often diplopia, her sight had become bad, and she had pain in and weakness of the L. side of her face.

State on Admission.

Special senses.—Smell and taste normal. Hearing, R. fair; L. lost. Sight, could only count fingers at a few feet. Intense optic neuritis.

C. N., 3, 4, 6.—No ocular palsy. Slight weakness of conjugate deviation of eyes to L. Unsteadiness of eyes on looking to R.; slow coarse nystagmus on looking to L.

No other cranial nerve affection.

Attitude.—Nothing noteworthy.

M. S.—Tone and strength of the muscles equal in the limbs of the two sides. A variable amount of tremor in the outstretched arms.

Co-ordination rather poor on both sides, but one arm was not definitely more ataxic than the other.

Gait.—Could walk alone fairly well, but staggered a little; did not deviate or stumble to either side, but seemed more uncertain on the L. than on the R. foot. If an attack of vertigo occurred when she walked, she rotated on her vertical axis to the L., and fell to the L. and backwards. Stood better on R. than on L. foot.

Sensory S.—No change.

Reflexes.—Deep, equal and brisk on the two sides. Superficial, normal. Plantars flexor.

Cranium.—No deformity or tenderness.

An attack of vertigo was observed a few days after her admission to hospital. It began with very severe headache, buzzing in

L. ear, and a feeling of coldness and numbness in the lower extremities. She at once rotated on her vertical axis to the L., and fell to the L. and backwards. Apparently there was no sensation of movement of external objects. Immediately after the attack both arms were very tremulous, there was considerable weakness of the L. side of her face and convergent strabismus, owing to paresis of the L. external rectus. Conjugate deviation of the eyes to the L. was also enfeebled.

Ten days after admission Sir Victor Horsley removed a firm ovoid tumour, measuring 4 by 2½ cms. from the L. ponto-cerebellar region. The patient died two days later from secondary hæmorrhage, but during the time she lived the deep reflexes of the L. limbs were absent, though those of the R. remained brisk.

Case 14.—*Tumour* (*glioma*), *left extra-cerebellar.*

H. C., aged 40 years, male. Under the care of Dr. Tooth. 1903.

Had syphilis 11 years previously. Health otherwise good.

Twelve months before he came under observation he had begun to suffer from severe occipital headache, which persisted. For the same time his L. ear had been becoming deaf, his memory was deteriorating, and he had been irritable and depressed. For six months had frequent attacks of giddiness, in which objects seemed to move in front of him from L. to R., generally no sense of movement of self. If standing, would fall forwards or to L. His gait had been affected for five months. He staggered, reeled, and deviated to the L. For three months had diplopia; found some difficulty in swallowing, and his speech had been slurred and indistinct.

State on Admission.

Special senses.—Smell normal. Taste good on R., almost absent on L. side of tongue. Hearing good in R. ear, lost in L. to both aerial and bony conduction. Vision natural; no optic neuritis.

C. N., 3, 4, 6.—Paresis of each external rectus, greater L. than R. Weakness of conjugate, lateral movement to both sides, but greater to L. On looking to R. fine rapid nystagmus, slower and coarser jerkings on looking to L. Vertical movements unimpaired, but on looking up there was rotatory nystagmus from R. to L. Pupils equal and reacted well.

5.—No masseter weakness, but considerable sensory loss on L. side of face.

7.—Slight weakness of the L. side of the face of peripheral type.

9, 10, 11.—Weakness of L. side of palate, difficulty in swallow-

ing solids, and fluids regurgitated through nose. Articulation nasal and slurred.

12.—Tongue protruded straight.

Attitude.—Nothing noteworthy.

M. S.—Muscles well developed, no rigidity or hypotonicity on either side, L. limbs considerably weaker than R. Co-ordination of movements of R. limbs fair, but considerable defect in the L. limbs, greater in arm than leg. Both arms tremulous when horizontally extended in front of him.

Gait.—Very unsteady and reeling, of the drunken type. Could scarcely walk alone. Constantly deviated to the L., saying "something seemed to draw him in that direction"; stumbled and fell to L. Stood more steadily on R. foot than on L. L. leg was held abducted, and rotated outwards.

Sensory S.—No change, save on L. side of face.

Reflexes.—All normal. Plantars flexor.

Cranium.—No bulging or local tenderness.

From the date of his admission into hospital he rapidly became worse, and suddenly died from respiratory failure.

At the autopsy a spherical tumour in part cystic lay on the L. side, between the pons and cerebellum, and excavating both considerably. It measured about 4 cms. in diameter. Microscopically it was a cystic glioma evidently of cerebellar origin, and largely degenerated in parts.

Case 15.—*Tumour* (*glioma*), *left extra-cerebellar.*

F. T., aged 32, male. Under the care of Dr. Ferrier. 1903.

His previous health had been excellent

For nine months he had suffered from severe headache, generally occipital, with pain in neck. Also occasional attacks of vomiting and vertigo, of the latter no definite description was obtained; progressive deafness of L. ear; numbness of L. side of his face and some difficulty in mastication were also of nine months' duration. For four months there had been some affection of articulation, slight difficulty in swallowing, and he had staggered and reeled like a drunken man when walking. For two months he had frequently diplopia and sight had failed.

State on Admission.

Special senses.—Smell normal. Taste lost on L. side of tongue. Hearing; R. slightly impaired; L. absent. Vision poor, intense optic neuritis.

C. N., 3, 4, 6.—Weakness of both external recti, greater on L.

Irregular nystagmus on horizontal movement, chiefly to L. Vertical movements normal.

5.—Weakness and wasting of L. masseter. Jaw deviated to L. Considerable loss of sensation over the whole L. trigeminal distribution.

7.—Slight L. weakness of the peripheral type.

9, 10, 11, 12.—Weakness of L. side of palate, swallowing difficult, regurgitation of fluids through nose. Articulation slurred and nasal. Tongue protruded to R.

Attitude.—Nothing noteworthy. Generally lay on R. side.

M. S.—Muscles well developed, their tone and power were about equal on the two sides. Slight inco-ordination of L. arm. Both hands tremulous.

Gait.—Very ataxic and unsteady; reeled and staggered about, stumbled and fell to L.

Sensory S.—No change except on L. side of face and mouth.

Reflexes.—Deep, brisk and equal on the two sides. Plantars flexor.

Cranium.—No bulging or local tenderness.

The patient's state gradually became worse from the time of his admission to hospital.

A month later, sensation was completely lost on the L. side of the face and mouth; there was greater weakness of conjugate deviation of the eyes to the L. and the R. external rectus was considerably weaker. Bilateral palate palsy and greater difficulty in swallowing and articulation also developed. The R. limbs had also become weaker than the L. and slightly rigid, and the deep reflexes were exaggerated in the R. The R. plantar reflex was constantly, and the L. occasionally, of the extensor type. His gait was so unsteady that he could scarcely walk alone; he was constantly stumbling and falling to L.

Four months after admission there was greater general weakness and emaciation. Swallowing was so difficult that he was generally fed by tube, articulation was quite unintelligible, and he was so drowsy that it was very difficult to examine him.

The R. limbs were spastic and weak, but the L. were fairly strong but rather flaccid and extremely inco-ordinate in movement. There was very noticeable forced rotation to the L., even when so weak that he could scarcely move voluntarily in bed, he was constantly rolling over to the L. side. The deep reflexes remained brisk on either side, but greater on R. than L., and the plantar reflexes were both extensor.

He died from respiratory failure.

At the autopsy a large tumour, mainly cystic, lay between and excavated the left lateral lobe of the cerebellum and the pons. It measured about 4 cms. in all diameters. Microscopically it was a cystic glioma of cerebellar origin.

Case 16.—*Tumour (glioma), left extra-cerebellar.*

W. T., aged 43, male. Under the care of Dr. Buzzard. 1904. Previous health good.

For four years he had had constantly "singing" in the L. ear with progressive deafness. Two years later began to have attacks of giddiness which gradually increased in frequency and severity. They consisted of a "feeling of weakness, dimness of vision, an indescribable sensation in his head, with nausea." No sensation of movement of self or of objects. During the short time the attacks lasted the patient was incapable of doing anything, though not unconscious.

About the same time he began to stagger and walk like a drunken man, but even later when his gait had become very unsteady he could ride well on horse-back. Two months previous to admission his vision began to fail, his hands became unsteady, and since then he has had constant frontal headache.

State on Admission.

Special Senses.—Smell and taste normal. Hearing—R. good; L. absent. Vision; considerable deterioration of both eyes. Very intense optic neuritis.

C. N., 3, 4, 6.—Slight weakness of L. external rectus and diplopia on looking to L. Nystagmus, coarse and deliberate on looking to L., more rapid and finer on looking to R.

5.—Slight hypoalgesia on L. side of face. No weakness or wasting of masseters.

7.—Some weakness of L. side of face of peripheral type.

9, 10, 11, 12.—No weakness of palate or tongue. No affection of swallowing or articulation.

Attitude.—No unnatural position of head. Lordosis as he stood.

M. S.—Tone and strength of muscles equal and good on the two sides. No hypotonicity or rigidity. Arms tremulous when horizontally extended. Marked inco-ordination of all movements of the L. limbs, greater in arm than leg, and similar but much less affection of the R.

Gait.—Considerable lordosis. L. shoulder held in front of the R. Feet rather widely separated. Reeled and staggered in a

very drunken fashion. Great tendency to deviate to the L. of the line of progression, and stumbled more to the L. than R. Could not stand on the L. foot alone.

Sensory S.—No affection of any form of sensation.

Reflexes.—Deep; brisker on R. than L. Abdominal absent on R. Plantar flexor responses on both sides.

Cranium.—Some tenderness in the occipital region.

The patient was operated upon a few days after admission by Sir Victor Horsley, and an irregular-shaped tumour (maximum diameter about 4 cms.) was removed from the L. posterior fossa. He recovered well from the operation, but died a few days later from septic infection, which probably resulted from opening the mastoid cells.

Case 17.—*Tumour (glioma), left extra-cerebellar.*

R. J., aged 46 years, male. Under the care of Dr. Hughlings Jackson. 1903.

Previous health had been excellent.

For two years he had been aware of gradually increasing deafness of L. ear, and more recently there was constantly a buzzing sound in it. The other symptoms were only of two months' duration. They were: attacks of giddiness in which he became blind and seemed to rotate on his vertical axis from R. to L.; in these attacks has often fallen to the L., unsteadiness in walking apart from giddiness, with a tendency to stumble to L. Intense pain in the occipital region, which radiated down neck. Numbness of L. side of face.

State on Admission.

Special senses.—Smell normal. Taste; good on R., absent on L. side of tongue. Hearing; R. normal, L. diminished, air and bone conduction. Vision good. No optic neuritis.

C. N., 3, 4, 6.—No squint or diplopia. Coarse, slow nystagmus on looking to L. and downwards, fine on looking to R. and up.

5.—Motor functions normal. Considerable anæsthesia on L. side of face.

7.—Paresis of L. side of face of peripheral type.

9, 10, 11, 12.—Palate; L. side weak. Tongue protruded slightly to R. No difficulty in swallowing or affection of speech.

Attitude.—Occiput approximated to L. shoulder, chin directed to R.

M. S.—Muscles well developed. Tone and power good on each side. No inco-ordination. Tremor of both outstretched hands.

Gait.—Walks in a drunken fashion with a constant tendency to deviate to the L. Stumbles and falls to L. No lordosis or other abnormality in attitude.

Sensory S.—No change, save on L. side of face.

Reflexes.—Deep; present and equal on the two sides. Superficial; normal.

Cranium.—No bulging or tenderness.

From the time he entered hospital the patient slowly became worse, and his gait was so much affected that six weeks later he could not walk alone, then slight improvement set in.

State Three Months after Admission.

Severe and persistent occipital headache. Optic neuritis had developed, and the attacks of giddiness were more frequent, in which he felt forced to roll over to the L. in bed, but never noticed movement of external objects. The L. ear had become completely deaf, but the hearing in the R. remained good.

C. N., 3, 4, 6.—Eyes constantly conjugately deviated to R. Weakness of L. external rectus. Nystagmus rapid and fine towards R., slow and coarse to L. Rotatory nystagmus to L. on looking up. No spontaneous nystagmus.

5, 7.—Facial anæsthesia and paresis increased.

9, 10, 11, 12.—Palate weak on L. Difficulty in swallowing and regurgitation of fluids through nose. Speech nasal and indistinct. Tongue protruded to R.

Attitude.—Occiput approximated to L. shoulder. Considerable lordosis when standing.

M. S.—Tone and power of muscles good and equal on the two sides. Great inco-ordination of L. limbs and slight irregularity of finer movements of R.

Gait.—Very ataxic. Could scarcely walk alone. Constantly deviated to L.; remarked spontaneously, "I am always going to the L.; I cannot help it." Stumbles and falls to L. L. shoulder held in front of, and higher than, the R., and L. leg abducted and rotated outwards. Stands more steadily on the R. than on the L. foot.

Sensory S.—Unaffected.

Reflexes.—Normal.

Operation.—A large bilobar tumour, measuring $3\frac{1}{2} \times 2\frac{1}{2}$ cms., was removed by Sir Victor Horsley from between the L. side of the pons and cerebellum. Immediate recovery from the operation was excellent, but the wound later became septic, and the patient died.

Case 18.—*Tumour, left extra-cerebellar.*

A. W., aged 42 years, male. Under the care of Sir William Gowers. 1904.

Previous to present illness he enjoyed excellent health. Eighteen months ago noticed some deafness in L. ear, which had been complete for nine months. Never tinnitus aurium. For a year daily attacks of vomiting; also headache always worse over the L. eye, but recently also occipital. For ten months very frequent attacks of vertigo; its subjective phenomena were: (1) false projection of objects, they always seemed to the R. and nearer to him than they were; (2) the floor would seem to rise up towards his face, but he could always locate it correctly with his feet; (3) the path on which he walked appeared "corrugated" for the next ten to twenty yards in front of him. Even though its surface was quite level he would for this space see it raised in a series of elevations, more or less regularly arranged, at distances of eight to ten feet from apex to apex. If he stood still there was no movement of the surface of the path, but as he walked "the waves undulated in front of him," keeping a constant distance from him and preserving their relation to one another. This phenomenon would appear and disappear quite suddenly and would recur for shorter or longer periods (from five seconds to five minutes); (4) if walking with a wall on his L. side the wall might appear similarly corrugated in the vertical plane; (5) objects would seem to move in front of him from L. to R. There was no subjective rotation of self, but he would walk in a "stumbling curve" to the L.

For eight months he had occasionally diplopia and his gait was affected. He staggered like a drunken man, but always deviated and stumbled to the L. During the three months before his admission his hands had been unsteady, his sight had failed and he had found some difficulty in swallowing and in articulation.

State on Admission.

Special senses.—Hearing; R. normal; L. absent. Vision, poor, intense optic neuritis.

C. N., 3, 4, 6.—No ocular palsy, but conjugate movement of both eyes to the L., weak and performed only with great effort. Slow, coarse nystagmus on looking to L., irregular and more intermittent to R. Also irregular vertical nystagmus on looking up and down.

5.—Slight anæsthesia on L. side of face and mouth. Masseters equally strong.

7.—Some weakness of L. side of face of peripheral type.

9, 10, 11, 12.—Functions normal.

Attitude.—Occiput always held approximated to L. shoulder. Lordosis as he stood.

M. S.—Development, strength and tone of muscles equally good on the two sides; considerable coarse tremor in both hands when held out in front of him. Some inco-ordination of the L. limbs, and in rapidly-repeated movements the L. arm does not move so freely as, and tires more quickly than, R.

Gait.—Varied very much, independently of vertigo. Sometimes needed assistance. A constant tendency to deviate to the L. and he staggered and fell to the same side. The actual deviation was greater than the apparent as he rotated his trunk on its vertical axis, L. shoulder in front of R., so that he faced to the R. of the line of progression. He recognised the tendency to deviate to the L., and occasionally, when walking fairly well, over-corrected it by this rotation of his trunk; then walked to the R. of the point he wished to reach. His basis was always broadened by abduction and outward rotation of his L. leg. Could stand on the R. foot alone, but not on the L.

Sensory S.—No change in any form of sensation.

Reflexes.—Deep and superficial equal on the two sides. Plantar flexor.

Cranium.—No bulging or tenderness.

Ten days after admission a tumour was removed by Sir Victor Horsley from the L. ponto-cerebellar region. It was spherical in shape and about 4 cms. in diameter, with a cyst placed on its upper surface. In its removal the L. auditory, facial, and trigeminal (sensory portion) nerves were destroyed.

The patient recovered slowly from the operation. After the first two weeks the deep reflexes were exaggerated, but R. more than L., and an extensor response was obtained in the R. foot. There was considerable weakness of the L. limbs, and they were very ataxic; the R. were only slightly unsteady. His state a month after operation was:—

Hearing.—R., normal; L., complete deafness.

C. N.—Great weakness of conjugate deviation of eyes to L., and slow, deliberate nystagmus on attempting to do so. Fine rapid nystagmus to R. Complete anæsthesia and paralysis of L. side of face.

M. S.—Left limbs very flaccid and hypotonic; no difference in strength. L. limbs very ataxic. Occasionally general tremulousness of head and limbs. Could not perform the pronation-supination with L. arm. No rotation movements or vertigo.

Sensory S.—No change.

Reflexes.—Equal and brisk on the two sides. Plantar—R. extensor, L. flexor.

Four months after operation the patient was considerably stronger. Flaccidity of L. limbs still definite, and they were very ataxic, though less so than formerly, but quite as strong as the R.

After six months began to walk alone, but fell, stumbled and deviated to L. Tremulousness disappeared and ataxia was less. No recurrence of vertigo in any form. Nystagmus still persisted.

Case 19.—*Tumour* (*Fibroma*), *left extra-cerebellar.*

W. M., aged 39, male. Under the care of Dr. Risien Russell, 1903.

Previous history unimportant.

For some years his walking had been unsteady and he had always kept some distance apart from his friends when walking, as otherwise he bumped into them. Eighteen months before admission into hospital he began to suffer from headache, vomiting and giddiness. The headache at first was frontal, but later became occipital and bilateral. The vomiting and giddiness were always present when the headache was severe.

During the last twelve months he became deaf in the L. ear, and his vision began to fail. Often diplopia and at times transient blindness. His speech became slow and indistinct.

State on Admission.

Smell normal. Taste defective on R. Hearing defective on L. Vision. — $\frac{6}{6}$ in both eyes, fields contracted. Intense optic neuritis.

C. N., 3, 4, 6.—Pupils equal, reacted normally. Weakness of L. external rectus. Lateral movements impaired to both sides. Nystagmus only occurred on lateral movements; fine and rapid to the R., slow and deliberate to the L.

5.—Slight affection of both motor and sensory divisions on the L.

7.—Slight weakness of the L. lower face.

9, 10, 11, 12.—Tongue flabby and tremulous, and was protruded to the L. Weakness of L. side of palate and of L. sternomastoid and trapezius.

Attitude.—The head was held slightly retracted with the occiput approximated to the L. shoulder; there was slight lordosis.

M. S.—Muscles were of equal size and good tone on both

sides, but the power of the L. limbs was considerably less than that of the R. Co-ordination was good on the R., but much impaired on the L. On extending the arms there was a considerable degree of coarse tremor, especially of the L. There were no spontaneous movements.

Gait.—Stood on a wide base with considerable lordosis and intermittent action of the spinal muscles. The L. shoulder was in front of the R. The L. leg abducted and rotated outwards, and its movements ataxic. Often walked towards the R., but invariably staggered and tended to fall to the L. He could stand on the R. leg for a short time, but not on the L. alone.

Sensory S.—No defect except that mentioned above (L. 5th).

Reflexes.—Deep, were all brisk, R. more than L. Epigastric and abdominal present, R. diminished. Plantar reflexes extensor. No sphincter trouble.

Ten days later Sir Victor Horsley removed a hard spherical tumour, measuring about 3½ cm. in diameter, from the L. posterior fossa, lying under the cerebellum near the seventh and eighth nerves on that side.

After the operation there was increase of the weakness of the L. sixth and seventh nerves, and for a time almost complete loss of conjugate movement of the eyes to L. Fine rapid nystagmus on looking to R., slower and coarser on attempting deviation to L. The patient was for some time quite blind. Articulation was slurred.

The power of the two sides was about equal, but the L. limbs were hypotonic and very ataxic. The tendon reflexes, which were lost after operation, returned in about two weeks and were eventually exaggerated, especially on L. side. Plantar reflexes flexor.

Patient continued to improve for two weeks, then became very irritable and mentally affected. This phase lasted about a month, then steady improvement set in and he became bright and intelligent.

Three months after operation the weakness of lateral movement of eyes to L. and the nystagmus were still present. Tone and power of limbs were good. Great ataxia of L. limbs, though movements of the R. were well co-ordinated. He could not execute rapidly-repeated movements with L. arm. He could walk with slight assistance. Sensation was unaffected, and the tendon-reflexes were brisk and equal on the two sides.

Seven months after operation he could walk alone with very little unsteadiness, but still some tendency to deviate and stumble to L. His vision had improved to $\frac{6}{6}$, coarse nystagmus on looking to L. but no other disturbance of cranial nerve functions.

Case 20.—*Tumour, right extra-cerebellar.*

C. T., aged 32 years, male. Under the care of Dr. Beevor. 1904.

Previous health and family history excellent.

For 16 months indefinite affection of gait. All the other symptoms were of 6 months' duration. Then began to walk like a drunken man, and became unable to turn quickly. Had frequently attacks of giddiness of which no definite description could be obtained, "things seemed to be mixed up anyhow," but there was never any sensation of movement of self. The vertigo as a rule only came on when he moved quickly, and the apparent movement of external objects was in the opposite direction to that in which he moved his head. In the attacks felt inclined to fall, generally backwards. For six months, too, the R. ear had been deaf; he had frequently diplopia and occasionally difficulty in swallowing. Had only had one short attack of headache and vomiting.

State on Admission.

Special senses.—Smell and taste good. Hearing, R. ear deaf to both aerial and bony conduction; L. natural. Vision poor; intense optic neuritis.

C. N., 3, 4, 6.—Slight weakness of R. external rectus. Slow coarse nystagmus was occasionally visible on looking to R.

5.—No affection of motor or sensory functions.

7.—Slight paresis in expressional and volitional movement of R. side.

9, 10, 11, 12.—Weakness of R. side of palate. Tongue protruded straight.

Speech, nasal. Slight difficulty in swallowing solids.

Attitude.—Nothing noteworthy.

M. S.—No difference between the tone of the muscles of the two sides, but the L. limbs were considerably weaker than the R. Co-ordination impaired on both sides, more so in L. than in R., and there was some intention-tremor on finer movements; also some coarse tremor in the horizontally-extended arms.

Gait.—Like that of a semi-drunken person, staggered, especially on turning. Constantly deviated to the R., but generally stumbled and fell to the L. Feet kept rather wide apart.

Sensory S.—Unaffected.

Reflexes.—Deep; all exaggerated on L., normal on R. Plantar extensor responses.

Cranium.—No local tenderness.

While in hospital his gait became more unsteady, the optic neuritis increased and his vision deteriorated.

State Five Weeks after he came under Observation.

Hearing.—R. quite lost; no tinnitus. L. good.

C. N., 3, 4, 6.—No ocular palsy observed. Nystagmus often accompanied lateral movement, but was not constant or regular. It most frequently resulted on looking to R., and was then always slow and forcible, being finer, rapid and intermittent on lateral movement to L. No nystagmus on vertical movement.

5.—No sensory loss on face, masseters equally strong.

7.—R. facial weakness, more marked in expressional than in volitional movement.

9, 10, 11, 12.—R. side of palate weak. Articulation nasal. Stridor during sleep, but vocal cords moved well. Tongue straight.

Attitude.—Occiput frequently approximated to R. shoulder. No lordosis.

M. S.—Tone of muscles about equal on the two sides. Some weakness of L. limbs resembling that of spastic hemiplegia, though the R. were not so strong as they had been. Tremor in the outstretched hands. Inco-ordination greater in R. than L. limbs, Babinski's pronation-supination test less well carried out L. than R.

Gait.—Walked on a broad basis with R. leg somewhat abducted. R. shoulder generally held in front of and higher than L. Deviated to R. but stumbled and fell to L. With feet together fell to either side.

Reflexes.—Deep; exaggerated on L., normal on R. Abdominal diminished on L. plantar; R. indefinite response, L. extensor.

Owing to the increase of the symptoms an attempt was made by Sir Victor Horsley to remove the tumour, but the patient died the evening after the first stage, *i.e.*, removal of the bone, was completed.

At the autopsy a large tumour, measuring $5\frac{1}{2} \times 4 \times 3\frac{1}{2}$ cms., was found lying between the pons and the under surface of the R. lateral lobe of the cerebellum. It excavated both, but did not directly involve either.

Case 21.—*Tumour* (*fibroma*), *left extra-cerebellar.*

J. J., aged 30; male. Under the care of Sir William Gowers. 1903.

The only noteworthy fact in the previous health of this case was a doubtful history of syphilis.

Three months before admission into hospital he had suffered

from severe headache, chiefly frontal; also diplopia, and for the last two weeks had been unable to walk, and became very drowsy, with occasional incontinence of urine.

State on Admission.

On admission he was dull and irritable.

Special senses.—Smell and taste were unimpaired. Hearing was diminished on the L. Vision—R. $\frac{6}{24}$; L. $\frac{6}{18}$; no optic neuritis.

C. N., 3, 4, 6.—Weakness of the L. external rectus, no nystagmus. Pupils equal and reacted well.

5.—Slight motor weakness of the L. trigeminus.

Other cranial nerves were normal.

Attitude.—He showed tendency to hold his head retracted.

M. S.—Power was good on both sides. No rigidity or flaccidity. Co-ordination was good.

Gait.—Uncertain, with a constant tendency to fall backwards.

Sensory S.—Was unaffected.

Reflexes.—Deep; all increased, R. more than L. Plantar reflex on R. side extensor; on L. flexor. Slight incontinence of urine.

His condition gradually became worse, and a month later was as follows:—.

Well-marked optic neuritis; L. ear deaf.

C. N., 3, 4, 6.—Weakness of L. external rectus; nystagmus on looking to L.

5.—Motor and sensory impairment of L. side very marked, with loss of taste on L. side of tongue.

7, 9, 12.—All weak on the R. side.

M. S.—Very little loss of power, but marked ataxia of both upper limbs, and to a less degree of the lower limbs, the L. being more affected than the R. There was no spontaneous tremor.

Reflexes.—Deep: all much increased, with ankle clonus on both sides, the increase being more on the R. than L. Plantar: R. extensor, L. indefinite. Incontinence continued.

Sensory.—No change except on face.

Cranium.—No bulging, tenderness in occipital regions on both sides.

Sir Victor Horsley removed a large growth from the L. posterior fossa a few days later. The patient recovered from the operation and improved steadily for over a fortnight; then he became dull and irritable, and refused his food. His temperature rose; he developed pneumonia and died semi-delirious.

At the autopsy it was found that a considerable portion of the tumour had not been removed; the remains lay in the L. surface

of pons excavating the ventral surface of cerebellum. Its greatest bulk lay at level of fifth nerve.

Case 22.—*Tumours* (*fibromata*), *extra-cerebellar, in each side.*

R. S., aged 16 years, male. Under the care of Dr. Taylor. 1903.

Always delicate. Cataract in R. eye at five years of age.

For two years had tinnitus in both ears, and gradually became deaf. Completely deaf in both ears for one year. Two years previously also began to stagger, he could not walk straight, gait gradually became worse, stumbled like a drunken man, but not more to one side than to the other. For three months the sight of his L. eye had been deteriorating, and he had had several transient attacks of blindness. For two months occasional headache, but no vomiting. No vertigo.

State on Admission.

Special senses.—Smell and taste normal. Hearing, deaf in each ear to both aerial and bone conduction. Sight—R. nil, opaque cataract; L. poor, intense optic neuritis.

C. N., 3, 4, 6.—Weakness of each external rectus, greater on L. than on R. Vertical movements and convergence unaffected. Nystagmus on lateral movement, more forcible in looking to L. than to R.

5, 7, 9, 10, 11, 12.—Functions normal, except that the L. side of the palate was weak, and the articulation slurred.

Attitude.—Head generally bent forwards. Trunk also bent forwards when standing, and stood on a broad basis like an old man.

M.S.—Tone and strength equally good on each side. Limbs rather unsteady when held out. Slight ataxia of both upper extremities, generally greater in L. than R. Rapidly-repeated movements fairly well done on either side.

Gait.—Walks very unsteadily, like a drunken man, with feet far apart. No marked tendency to deviate to either side, bu stumbles to both, chiefly to L.

Sensory S.—No affection.

Reflexes.—Equal and brisk on the two sides. Plantar responses flexor.

Cranium.—Some bulging of L. occipital region. No tenderness.

During the next two months the patient became weaker, and his gait much more ataxic. Sometimes the inco-ordination was more marked in the L., sometimes in the R. limbs.

He was then operated upon by Sir Victor Horsley, and an

irregular ovoid tumour about 4 cms. in diameter was removed from the L. posterior fossa; considerable injury to that lobe of the cerebellum resulting, as the growth was taken out through it. For some hours after the operation well marked "skew-deviation" of the eyes, the L. being directed down and inwards, the R. slightly up and outwards.

The patient recovered fairly rapidly from the operation.

The deep reflexes were for a time diminished in the L. limbs, but soon became brisk and equal. For some weeks there was considerable weakness and flaccidity of the L. limbs, and they were very ataxic in their movements.

State Six Weeks after Operation.

Hearing.—Quite deaf in each ear. Sight $\frac{6}{36}$, optic neuritis subsided into atrophy.

C. N.—Weakness of L. external rectus, coarse nystagmus on looking to R. and L., more regular and deliberate in latter direction.

Considerable paresis of L. side of face, of peripheral type.

Articulation nasal and indistinct, but no paresis of palate.

Attitude.—Generally bent forwards. Occiput approximated to L. shoulder.

M. S.—Muscles hypotonic, especially those of L. limbs, which are much weaker than R. Ataxia of limbs of two sides, much greater L. than R. Hands very tremulous when held out in front of him.

Gait.—Could only walk a few steps alone, deviated to L., and reeled and stumbled in same direction. Fell always to L.

Sensory S.—No change detected.

Reflexes.—Equal and normal in type on the two sides.

The patient's condition remained more or less unchanged while he was under observation. After several months he became very drowsy and lost the power of walking. He died at home eleven months after the operation.

Autopsy.—The greater part of the L. lateral lobe of the cerebellum was destroyed, and a collection of cerebro-spinal fluid separated its remains from the dura mater. A large spherical firm tumour lay in the R. posterior fossa compressing the pons and cerebellum on that side. It was loosely adherent to the membranes.

Reviews.

Les Psychonévroses et leur traitement moral. (Leçons faites à l'Université de Berne.) Par le Dr. DUBOIS, Professeur de Neuropathologie. Préface du Dr. DEJERINE). Paris, Masson et Cie., 1904. Pp. 557.

THIS is a book of considerable interest. It is obviously written by a physician of experience and judgment, who has thought out for himself the problems presented to him by his patients. The advice given is, speaking generally, excellent and marked by common sense. Unfortunately the author has not learned the ar of condensation, and the book consequently suffers from being too long. It contains a number of accounts of cases, given in a conversational manner, with reports of the colloquies that took place between the doctor and patient. The reports are well told and often witty, and, from the author's point of view, illustrate his method of treatment, in which advice and moral direction play the principal part, but, on the other hand, they are somewhat vague as regards clinical signs and symptoms, and the reader has often to take the diagnosis for granted. Essentially the book is a development and expansion of the theme that the most important part of the treatment of the various neuroses lies in the moral influence of the physician over the patient, and that success in treatment is to be attained, with certain minor adjuvants, by exerting this moral force or domination. This is, of course, by no means a new discovery, and the author is far from claiming it as such, but he thinks that up to the present time its value has not been fully utilised, although one would have thought that the influence of the mind on the body has been pretty much in the air of late, and that the physician's attention is too frequently drawn away from this, which he regards as the only permanent method of cure, to the passing fashion in treatment, whatever it be—hypnotism, electric baths, the high-frequency current, or radiotherapy. No one would dispute the original proposition that by force of character or will-power, to use a popular term, the physician may often, if he can get the patient placed under favourable conditions, achieve wonders in the way of cure of the neuroses. Indeed, re-

garded from this point of view, a large part of all medical treatment is a kind of suggestion. We must, however, be careful in talking of suggestion, for the author prefers the word persuasion or moral suasion to describe his method, avoiding the word suggestion because it is mostly used in a bad sense. He quotes von Bechterew's saying that suggestion comes in by the back stairs, whilst persuasion enters at the front door. He, quite rightly, protests against the employment of hypnotic suggestion, as not justifiable in the interests of the patients, often, indeed, constituting a kind of moral vivisection. He would reserve hypnotic suggestion for exceptional use with due precaution in certain obstinate cases of hysteria, and generally substitute for it moral suasion. With moral suasion he combines where necessary other modes of treatment, and seems mostly to employ in suitably-selected cases the Weir-Mitchell cure, with its main factors of isolation, rest, over-feeding, and massage, varying the use of each of these means according to individual needs. As showing a minor point of difference between the two countries, little or no stress is laid upon the importance of ensuring a suitable nurse for the cure, indeed, judging from one or two hints, and also from the statement that the conversational powers of the masseuse afford a pleasant relief to the monotony of isolation, the inference is that a special nurse is at any rate not always employed.

There is, of course, no doubt of the influence that a doctor of forceful character may obtain over his patient, and especially over a hysterical or neurasthenic one; but, unfortunately, this is just one of the qualities of a doctor that "is born and not made," and cannot be imparted by much taking of thought. At the same time a book of this kind may be read with profit, especially by a man just beginning practice; for although, in the absence of natural aptitude, it cannot give him the art, it can be of great help in aiding him to learn how to advise and to treat his neurotic patients.

There is another aspect of the moral influence of the doctor over his patients, of which readers of Oliver Wendell Holmes' "Elsie Venner" will not need to be reminded, and that is, that in patients of the opposite sex this influence, good in its first effects, may ultimately become prejudicial to their welfare, and lead to unforeseen difficulties.

To give a further account of the book, it opens with the discussion of the psychological problems which underlie such a system of psychotherapeutics. We soon find ourselves, like our first parents in the Garden—and we may say, in passing,

that the first recorded instance of suggestion occurred there—engaged in a discussion on free-will and determinism, with a passing glance at predestination. It is needless to remark that the conclusions arrived at are not those of Milton. The chief symptoms of neurasthenia next receive consideration, the leading characteristic being held to be readiness to experience fatigue (*fatigabilité*). The author deems it unnecessary for his purpose to describe the symptoms of hysteria, which he regards, with certain exceptions, as *une maladie psychique par excellence*, as *ideogenic*. He would class both diseases together as different manifestations of a general state of nervousness (nervosisme). It is difficult, however, to see the advantage of so doing. No doubt the present views of these disorders will undergo considerable modification in the future, but advance rather lies in the contrary direction of further and more exact differentiation.

The author makes some sound observations on the necessity of treating the general state in the neuroses; too often in such cases the patient goes from one specialist to another, according as the symptoms vary from time to time. "One is neurasthenic," according to him, "just as one is lazy, tired, or irritable." The chapter on sexual disorders conveys sensible advice, with a caution against a too elaborate investigation of the patient's sexual history. The value, at times, in both hysteria and neurasthenia, of a studied neglect of symptoms in promoting their disappearance, whilst at the same time the patients are unconsciously helped on to a better state of mind, is insisted upon in several places.

In the last chapter Dr. Dubois gives a summary of his position. For the prevention of the neuroses he looks with greatest hope to the cultivation of the intellectual and moral faculties, rather than to the development of the body. The present rage for athletics finds no support from him; he remarks that the latter-day equivalent for *mens sana in corpore sano* seems to be that the cultivation of the body necessarily *entails* a sound mind. In this age of hurry he insists on the necessity for a due amount of repose. The maxims of Seneca are several times quoted and recommended to patients for their inward digestion; and the author's ideal of life seems to be a genial stoicism to carry through the difficulties of the hour.

In conclusion, the author writes well, and although one may not agree with all his views, and may possibly think that the leading idea is sometimes pushed somewhat far, yet his advice is marked

by original thought, sound sense, and is evidently based on a considerable experience. There is, however, much repetition, and the book would gain in value by a certain amount of condensation.

J. MICHELL CLARKE.

Organic Nervous Diseases. BY M. ALLEN STARR, M.D., LL.D. Baillière, Tindall & Cox, London, 1903.

THIS is a book of great merit. It combines in its 700 pages a synopsis of current knowledge with the results of years of observation by a most able and experienced clinician. For this reason the book is more than a text-book, and may be regarded as a valuable contribution to neurological literature. Perhaps for the same reason it is slightly unequal in accordance with the interest taken by the author in one or other subject. Nothing, for instance, could be better than the chapters dealing with diseases of the peripheral nerves, with diagnosis and location of diseases of the spinal cord or of the brain, and they stand in marked contrast with the description of the symptomatology of tumours of the cerebellum. It would need considerable good fortune for anyone to make a correct diagnosis of the presence of a cerebellar tumour, or to state whether it was situated in the right or left, or middle lobe, from the information supplied in the page and a half devoted to the subject. The absence of any mention of hemiparesis, of inco-ordination apart from that suggested by the word "staggering," or of the diagnostic value of head positions, are noteworthy.

An Englishman will hardly be likely to agree with the statement that multiple sclerosis is a very rare affection, but th author is not alone among his countrymen in affirming the infrequency of the disease in America. At the same time, it is rather a matter of regret, from the students' point of view, that the cases cited in this connection are so atypical, and have not been the subject of pathological verification. In spite of a few minor defects, the book is one which may be confidently recommended to neurologists, not only on account of its originality and the general excellence of the descriptions, but also for the valuable hints on treatment which it provides and for the beautiful pathological and clinical illustrations, which are more numerous and better reproduced than in any text-book on the subject with which we are acquainted.

E. FARQUHAR BUZZARD.

Contributions from the William Pepper Laboratory of Clinical Medicine. (University of Pennsylvania. No. 4, 1903, Philadelphia.)

THIS volume contains twenty-nine articles which are either reprints from papers already published or read before various societies. Sixteen of them deal with the nervous system, and of these four are by Dr. William G. Spiller, who also participates in seven others. It is not possible to notice all the valuable papers collected in this volume.

The first paper is by Dr. Spiller, and describes the results of unilateral degeneration of the glossopharyngeal and vagus nerves in a case of fracture of skull.

A man, aged 54, fell and fractured his left occipital bone into the foramen magnum—bony fragments pressing upon the nerves of the medulla. The patient lived for forty-six days. The essential points in the clinical history were complete paralysis of the left half of the larynx, and complete inability to swallow. As the patient declined to be fed by the nasal tube, rectal alimentation had to be employed.

At the autopsy an area of hæmorrhage and softening was found on the under surface of the cerebellum. The roots of the left glossopharyngeal and vagus nerves were intensely degenerated. A point of great interest was that the sterno-mastoid and trapezius muscles were not paralysed, showing that the association of the spinal accessory nerve with the lower vagus roots within the jugular foramen cannot be very intimate.

The left lung was congested and contained numerous areas of consolidation, some breaking down into abscesses the size of a split pea. The right lung showed a similar condition confined to the middle lobe. This bronchopneumonic condition is not uncommonly observed after injury of the vagus. From the fact that the taste buds had disappeared from the circumvallate papillæ on the left side of the tongue, the author concludes that they must be innervated by the glossopharyngeal nerve, and he quotes in support a case of Rosenberg's in which a similar degeneration occurred in a man in whom the ninth nerve had been destroyed by a tumour. Observations on the condition of the pulse were too scanty to be of any use.

Dr. Spiller also gives an account of the pathological changes in the nervous system in a case of lead poisoning.

The patient was a man, aged 48, who had worked in lead for twenty years. He had had attacks of lead colic; a charac-

teristic blue line was present on the gums; slight wrist drop was present with complete paralysis of legs, incontinence of urine and delirium. While in hospital the paralysis of the lower limbs largely disappeared, while that in the upper extremities became more marked. No mention is made of alcohol as a possible additional factor.

The case, therefore, was one eminently suitable for examination.

The anterior cornual cells of the cervical and lumbar regions were intensely degenerated; the lumbar posterior roots were little altered. The median nerve showed marked degeneration by Marchi and Weigert hæmatoxylin stain, and the extensor muscles of the forearm showed much degeneration of nerve bundles within the muscles. In the brain, the chief change noticed was proliferation of the endothelial cells on the outer surface of the pia.

The other papers are mainly reports of interesting cases, with one experimental research on regeneration of posterior spinal roots. Among the papers not relating to the nervous system, two are of especial interest, one a careful report of a case of chronic acetanilid poisoning with marked alterations in the blood, by Alfred Stengel and C. Y. White; the other, a discussion on methods of determining blood pressure in man, by W. B. Stanton.

There are numerous illustrations throughout the volume, and altogether it does great credit to the laboratory from which it emanates.

A. E. Russell.

Vorlesungen über den Bau der Nervösen Zentralorgane des Menschen und der Thiere. By Dr. Ludwig Edinger. Erster Band, "Das Zentralnervensystems des Menschen und der Säugethiere." Seventh Edition, Leipzig, 1904.

The seventh edition of Dr. Edinger's well-known work is to be published now in two volumes. The first volume here reviewed deals with the central nervous organs of men and of mammals, and the author has made many alterations rendering this volume still more valuable than its predecessors to the physician and the pathologist, which embody the results not only of pure anatomical research, but also of pathological and experimental investigation.

In order to facilitate topographical comprehension of transverse sections of the nervous system, the pictures of the transverse

section appear as the anterior or posterior surface of the nervous system cut transversely and drawn in relief. The relation of internal structure to the external conformity, always a matter of difficulty to the student, is clear from the first glance at these admirable pictures. The number of these illustrations in relief is not so extensive as the student of serial sections might wish, but each subdivision of the brain stem and spinal cord is figured.

The paucity or absence of explanatory lettering to some of the illustrations both upon the plates and in the text, while it adds to the clearness and artistic effect, necessitates a careful reading of the context.

The first four chapters deal with the methods of investigating the anatomy of the nervous system, and with the general structure, physiology and development of nervous organs. An historical survey of the chief workers in this branch of science from the time of Vesalius to the present day is given, and the methods of research used by these pioneers is briefly described. In the description of the neuroglia, the author inclines strongly to the opinion that in diseases of the nervous system glial proliferation is always secondary to degeneration of the nerve elements. The present state of our knowledge with regard to the neuron hypothesis is clearly laid out. Great importance is attached to the existence in post-embryonic life of incompletely developed nervous paths. The attainment of physical and mental culture by practice is accompanied by the opening up of such paths and the complete medullation of their fibres. The capacity for anatomical development in the central nervous system persists throughout life as long as the capacity for learning remains.

The special anatomy of the mammalian central nervous organs is described in the form of nineteen lectures, the human brain being taken as the type. The comparative development of the human brain is first dealt with, and the relations of its parts to those of the brains of lower animals is made beautifully clear by the superposition of the outlines of the mammalian brain in red upon a black relief drawing of a Teleostean brain.

The macroscopic anatomy of the brain and spinal cord is most lucidly described, the omission and inclusion of details being selective with regard to their physiological and pathological utility.

The description of the segmental innervation of the skin and of the muscles has been previously published by Dr. Edinger. It is much at variance both with the opinions of the highest authorities, and with clinical facts. It has been put forward as tentative

only, but even at that it is not in keeping with the high standard of excellence of the book. The region of the umbilicus is certainly supplied from the ninth and not from the tenth dorsal roots. Such areas as are represented for the segmental skin supply of the legs are never seen clinically. On the other hand, the prolongations upwards of the L 3, L 4, S 1, S 2 areas, are probably the correct solutions of a much-debated question. The meeting of the area C 2 with the area of the fifth nerve in front of the angle of the jaw as figured, must surely be a mistake in the lettering, for it is contradicted by the area marked C 2 upon the occipital region.

The structure of the spinal cord is described with great clearness, and without any superfluous detail. Dr. Edinger's description of the ending of the pyramidal tracts in the spinal cord leaves the student erroneously convinced, and the critic entirely unconvinced. He states that the fibres of the pyramidal tracts end in the region of the ventral horn cells. Those of the ventral pyramidal tract can readily be traced into the ventral horns, where they can be seen breaking up. But the collaterals from the lateral pyramidal tracts cannot be traced into the ventral horns by the Marchi method in the human adult, but can only be followed thither in the embryo, and by the "silver" method. No mention is made of the conspicuous ending of the lateral pyramidal collaterals around the cells of Clarke's column. To argue a similar ending for the ventral and lateral pyramidal fibres, when the former can always be demonstrated and the latter never, is far from conclusive, while to ignore the accumulation of evidence of the highest authorities, that the lateral pyramidal collaterals can only be traced to the region of Clarke's column, is not justifiable.

Fig. 71, page 129, is a diagram representing the various tracts which may present in a transverse section of the cord. Taking a pre-pyramidal position in the lateral column, a large area is marked "tractus thalamo-spinalis." So conspicuous is this large tract upon a most important diagram that it is most disappointing to find no reference to it in the text. The opinion that the loss of pain sense, and of temperature sense, is to be connected with disease of the gray substance of the posterior horns is of great interest.

In the description of the medulla oblongata and pons, Dr. Edinger introduces some innovations of terminology which are most useful. The large system of descending fibres which occupies the middle third of the mesial fillet, and lies between the cortico-bulbar tract (mesial) and the chief sensory path (lateral), and which passes with the fillet fibres through the interolivary layer

and ends in the lower medulla, is named the "tractus thalamo-bulbaris"; while the fibres which reach the ventro-lateral region of the cord constitute a "tractus thalamo-spinalis." The so-called juxta-restiform body is named "tractus nucleo-cerebellaris." Fibres passing directly to the vermis cerebelli from the vestibular nerve and from the fifth nerve constitute the "direct sensory cerebellar roots" of these nerves. A tract, the fibres of which arise throughout the sensory nucleus of the fifth nerve, and passing directly across the middle line, collect upon the opposite side in a position lying between the descending root of the fifth and the central tegmental tract to ascend to the thalamus, is named the "tractus quinto-thalamicus."

Of especial interest in the description is the nervous mechanism subserving taste. The terminal nucleus of taste is a mass of gray matter which lies mesial and somewhat dorsal to the restiform body in close connection with the fasciculus solitarius of which it forms the end nucleus. Reaching from the upper cervical cord to the middle of the pons, this nucleus receives fibres from the back of the tongue, &c., *via* the glossopharyngeal nerve and petrous ganglion, and from the front of the tongue (1) *via* the chorda tympani geniculate ganglion and portio intermedia, and (2) *via* the lingual and Gasserian ganglion. This seems to be a happy solution of a much disputed question.

Hellweg's triangle is described as an ascending tract under the name of tract spino-olivaris. The cells of Deiter's nucleus are in close synaptic connection with collaterals of the vestibular nerve, and from these cells arise two most important sets of fibres: (1) the vestibulo-spinal tract; (2) fibres which, crossing to the dorsal longitudinal bundle of the same side and of the opposite side, divide into two, the upper prolongation passing *via* the dorsal longitudinal bundle to the motor nucleus, and the lower passing in the dorsal longitudinal bundle to the ventral column of the cord. This description and the accompanying diagram, fig. 108, will serve to illustrate the clearness and brevity with which the author impresses matters of great fundamental importance upon the student.

The mesial fillet is definitely divided into three groups of fibres—a lateral group, the chief sensory path; a central group, the thalamo-bulbar tract (descending system of fillet); and a mesial group consisting of the pyramidal projection for the motor cranial nerve nuclei.

After stating that the pyramidal projection for the motor cranial nerve nuclei descends in the two positions: (1) by the

accessory fillet, and (2) by the mesial fibres of the ventral pyramid, Dr. Edinger inclines to Dejerine's view, that Spitzka's bundle may be a third path for these fibres, and is possibly identical with the accessory fillet, the bundle being sometimes placed dorso-laterally—Schlesinger's accessory fillet, and sometimes mesio-ventrally—Spitzka's bundle. This opinion does not appear justifiable in the light of the work of Schlesinger, Hoche and Barnes.

The morphological description of the cerebellum is modified according to the recent work of Elliott Smith.

In the diagram representing the connections of the superior peduncle, the lateral bundle of Probst, which passes the red nucleus to end in the ventral nucleus of the thalamus, is figured.

The statement that " the symptoms which result from disease of the lateral lobes of the cerebellum are still quite unknown," will not appeal to those who are accustomed to diagnose with accuracy lesions of the lateral regions of the cerebellum by means of the signs which Risien Russell has laid down as symptomatic of such lesions.

To facilitate the comprehension of the symptoms resulting from lesions of the brain stem and thalamus, several illustrations are provided of transverse sections of these regions upon which supposed lesions in various positions are depicted as shaded circles, and the symptoms which resulted from a lesion in each position in actual cases is appended.

The central auditory path is figured as decussating completely in the corpus trapezoides, a semi-decussation of auditory fibres does not occur, and the central acoustic path seems to be in connection with the opposite ear only. The clinical fact, that lesions destroying the lateral fillet or the posterior colliculus do not cause complete deafness of the opposite ear, has therefore no anatomical explanation at present. The descriptions of the oculo-motor mechanism, and of the complex of the dorsal longitudinal bundle, are of particular excellence.

It is of special interest that degenerate fibres can never be traced into the oculo-motor nuclei after destruction of those cortical centres from which ocular movements are readily elicited by stimulation.

The morphology of the cerebral hemisphere is admirably described, Elliott Smith's new terminology being adopted.

The genetically older part of the pallium, the archipallium, consists of a basal portion (olfactory lobe) and a marginal portion (hippocampal formation). The remaining portions of the hemispheres which are of later origin constitute the neopallium, and

these first make their appearance between the basal and marginal portions of the archipallium. The anterior commissure is the chief commissure between the lateral halves of the archipallium, the psalterium being a second archipallial commissure. With the development of the neopallium and its large commissure (the corpus callosum), the marginal portion of the archipallium and its commissures (the hippocampal formation and the psalterium) are pressed backwards, and come to occupy a ventro-caudal position, the limbic fissure marking the limit between the archipallium and the neopallium.

The volume closes with an excellent résumé of the subject matter of the second part. The present edition of the work embodies the results of the most recent researches, and it is entitled to be regarded, even more than its predecessors, as a text-book for students quite unrivalled in any language. The text is a model of clearness and of classic German. The illustrations are abundant and beautifully reproduced.

The prominence given both in illustrations and in the text to the works of recent English authors, and especially to those of Dr. Elliott Smith, will be highly gratifying to English readers.

JAMES COLLIER.

L'Epilessia. By P. PINI. (Milan, Ulrico Hoepli, 1904.)

THIS small volume is a résumé of recent work on the subject of epilepsy, and, while not adding anything original, is certainly a full representation of the present state of our knowledge, especially of treatment.

The plan of the book is to discuss first the various theories as to the causation of epilepsy, to examine fully the "fluids," urine, sweat, blood, cerebro-spinal fluid and the gastric juice. According to the finding in each case, so are epileptics to be treated. Thus, diminution of the alkalinity of the blood calls for treatment that will raise the reaction to standard. The next theory discussed is the bacteriological, and the author suggests that at some future time epileptic seizures may be amenable to "serum" treatment. In support of this, he mentions cases which were free from attacks during the course of erysipelas. The greater portion of the book is occupied by a dissertation on the various methods of treatment by different forms of bromides, biborate, hypnotism and baths. Lastly, an account is given of epileptic colonies. Our opinion of the book is that it will prove a useful monograph for the author's countrymen. We cannot say it supplies a want in international medical literature.

DOUGLAS STANLEY.

La Nevrasthenia. By Dr. LUIGI CAPPELLETTI. (Milan, Ulrico Hoepli, 1904.)

THE prevalence of neurasthenia in modern life is ample apology for the appearance of any fairly good work on the subject. We believe that Dr. Cappelletti's book is a very complete description of the leading characteristics of the disease, and quite equal to the more usually read accounts of neurasthenia in English. The order followed is convenient, the author giving a general description of the symptoms and etiology, next a minute account of the special senses, general sensation, the respiratory, circulatory, and other systems. The importance of recognising the underlying condition of neurasthenia in some forms of cardiac and gastro-intestinal diseases is of course great, but we notice a tendency on the part of some writers on this subject to attribute to neurasthenia many forms of gastric disease, forgetting that such disease may have induced a general neurasthenic condition. Thus, defects of the pylorus and "coprostasis" are more likely to cause than to be caused by neurasthenia. The present work is open to this criticism. The writer devotes a chapter to the consideration of the leading theories of neurasthenia, such as Bouchard's auto-intoxication, Beard's nervous theory, ending with a criticism of them all.

By far the most thorough part of the book is that dealing with treatment; here the more practical methods of dealing with the neurasthenic state are detailed, such as massage, electricity, baths, Weir-Mitchell's method, and others. In conclusion, we are of opinion that, as a monograph, Cappelletti's work is useful and comprehensive, and a valuable addition to Italian medical literature.

DOUGLAS STANLEY.

Annali della Clinica delle Malattie Mentali e Nervose della R. Università di Palermo. Vol. ii. (Palermo, Fratelli Marsala-Editori, Angolo Via Parlamento.)

THIS collection of monographs appearing together is a variable record of researches carried on at Palermo University for two years. The volume contains a variety of papers, among which must be mentioned "A Study of Tubercular Neuritis." The article is by Colella, and shows evidence of considerable labour. We require further evidence of the occurrence of neuritis in tuberculosis, as in many cases it has not been proved that alcoholism has been altogether excluded. Some of the cases

quoted by the writer appear genuine, if we may rely on the history, but, as is well known, patients' statements require to be corroborated. Accompanying the paper are several very carefully executed plates, showing segmentation of the white sheaths, pigmentary infiltration of the cornual cells, vacuolation of nuclei, and the usual lesions found in neuritis occurring under other circumstances. The same writer contributes a paper on "Traumatic Psycho-neuroses," which is interesting and instructive. Another paper of value is "An Investigation into the Effects of Electricity on the Cerebral Pulse," by Emanuele Gentile, in which the writer describes the effect on the amplitude and duration of pulsation in the cerebral vessels when stimulated by faradic and galvanic currents, the latter in different directions. A carefully illustrated paper on the structure of nerve cells is contributed by Messina-Vitrano, based on the methods of Donagio, Cajal and Golgi. Such a collection of papers is a valuable addition to any reference library.

DOUGLAS STANLEY.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

A PATHOLOGICAL MEETING was held on Thursday, December 1, 1904, at 8.30 p.m., at 11 Chandos Street, Cavendish Square.

A discussion on "Lesions of the Posterior Columns of the Spinal Cord in Diseases other than Tabes Dorsalis" was opened by Dr. David Orr.

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EDITOR.

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A JOURNAL OF NEUROLOGY,

FOR

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AND

LIST OF OFFICE BEARERS AND MEMBERS

OF THE

Neurological Society of the United Kingdom,

1904,

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1903.

London:
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83-91, GREAT TITCHFIELD STREET, OXFORD STREET, W.
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Neurological Society of the United Kingdom.

REPORT OF THE COUNCIL FOR 1903.

FEBRUARY 4, 1904.

The Council has much pleasure in reporting the continued increase in the prosperity of the Society, which now numbers 246 members.

It regrets the loss of its honorary member, Mr. HERBERT SPENCER, who died on December 9, 1903, and of its corresponding member, GEHEIMRATH PROFESSOR JOLLY, who died on January 6, 1904.

The Presidential Address given on January 22 by Professor J. N. LANGLEY, F.R.S., on "The Autonomic Nervous System," was fully reported in the Spring number of "Brain." The abstract of the Proceedings of the Society shows the scope and character of its work during the past year. A new feature will be found in the dedication of a whole meeting of the Society to a single subject in Neurology, illustrated by typical and by unusual cases. The meeting devoted to Myopathy in its various forms was a great and encouraging success.

In consequence of the untiring energy and hospitality of the Edinburgh members, the meeting of the Society in that city will remain a most pleasant memory to all who were fortunate enough to be present.

On November 5 the third Hughlings Jackson Lecture was delivered by Sir WILLIAM BROADBENT, Bt., F.R.S.

The outstanding accounts for printing, publishing, and illustrating "Brain" have been a severe strain on the limited funds of the Society, but, in consequence of the care of the Editor and the persistent efforts of the Treasurer, the Society is now again entirely free from debt. Moreover, in addition to a small balance, the remaining copies of the Index to "Brain" are a valuable asset.

It is hoped that the prosperity of the Society will continue to increase, and so enable it to maintain the present high reputation of its Journal "Brain."

Proceedings of the Society during the Year 1903.

January 22.—Dr. J. N. LANGLEY, Inaugural Address, "The autonomic nervous system."

February 26.—Clinical Meeting; Subject, "Myopathy." This meeting was devoted to the exhibition of cases of idiopathic muscular atrophy.

April 2.—Dr. FARQUHAR BUZZARD, "The pathology and bacteriology of Landry's paralysis."

June 27.—Meeting of the Society at Edinburgh.

October 22.—Clinical meeting. Dr. HARRIS and Mr. LOW, "Case of Erb-Duchenne paralysis treated by operation." Dr. T. GRAINGER STEWART, "A case of diplegia and myoclonus." Dr. LEONARD WILLIAMS, "A case of infantile hemiplegia of the right side, with paralysis agitans beginning on the left side." Sir VICTOR HORSLEY, "Three cases of cerebellar tumour after operation, with reference to the question of cerebellar compensation." Dr. DUNDAS GRANT, "Three cases of nervous lesion, with manifestations in the throat and ear." Dr. GORDON HOLMES, "Three cases of unilateral tremor in cerebral lesions." Dr. COLLIER, "A case of (?) disseminated sclerosis." Dr. F. E. BATTEN, "A case of (?)." Dr. FARQUHAR BUZZARD, "Progressive muscular atrophy in a young woman."

November 5.—Hughlings Jackson Lecture, delivered by Sir WILLIAM BROADBENT, "Hughlings Jackson as pioneer in nervous physiology and pathology."

December 3.—Dr. FARQUHAR BUZZARD, (1) "Descending spinal fibres of a lumbar posterior root"; (2) "A dermoid tumour of the brain." Dr. MICHELL CLARKE, (1) "Diphtheritic paralysis"; (2) "Gumma of the spinal cord." Dr. H. A. CALEY, "Hydatid of the brain." Dr. J. S. COLLIER, (1) "Fibres of the pyramidal tract ending in Clarke's column"; (2) "Ending of fibres of the sacral triangle"; (3) "Specimens from cases of cerebral diplegia." Dr. W. HARRIS, "Prefixed and postfixed types of brachial plexus." Dr. JAMES FLASHMAN, "Advantages of the clove oil celloidin embedding method." Dr. FLETCHER and Dr. BATTEN, "The pathology of a case of progressive muscular atrophy in a child aged 11."

BALANCE SHEET, 1903.

Dr. NEUROLOGICAL SOCIETY. **Cr.**

RECEIPTS.	£	s.	d.	EXPENDITURE.	£	s.	d.
To Balance	79	10	2	By Messrs. Macmillan for publishing "Brain," 1902 ...	131	17	3
Subscriptions at £1	15	0	0	,, ,, ,, ,, 1903 ...	128	14	0
,, at £1 1s. (less 8s. not received) ...	247	8	7	Stationery and Printing—Bale and Danielsson ...	26	18	2
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Contribution of Messrs. Macmillan towards cost of Illustrations for 1902	4	6	11	,, Bale and Danielsson ... £14 19 0	34	19	0
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Contribution of Messrs. Macmillan towards cost of Illustrations for 1903	12	15	2	Lantern—Hawes	1	10	0
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				Medical Society £21 15 0			
				University College £1 5 0	23	0	0
				Bank Charges	0	4	0
				Balance	62	0	10
	£414	11	9		£414	11	9

Account for Publishing Index to "Brain," Vols. I.—XXIII.

	£	s.	d.		£	s.	d.
To Sale of 51 Copies	15	4	5	By Messrs. Macmillan's Account for 1902	68	8	8
Balance against	54	10	0	,, ,, Commission on 51 Copies ...	1	5	9
	£69	14	5		£69	14	5

General Statement.

	£	s.	d.		£	s.	d.
To Balance on General Account	62	0	10	By Messrs. Macmillan on Index Account	54	10	0
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Audited and found correct, December 21, 1903 { H. LEWIS JONES. E. FARQUHAR BUZZARD.

CHARLES E. BEEVOR, *Treasurer.*

RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Guinea, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due, shall be considered as equivalent to resignation.

15.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, the Editor of "Brain," and twelve Councillors, of whom not less than three shall be extra-Metropolitan Members.

16.—Five Members of the Council shall form a quorum.

17.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

18.—The office of Vice-President shall be tenable for two years, one Vice-President being elected every year.

19.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

20.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

21.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

22.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

23.—There shall be six Ordinary Meetings annually, of which the first held in each year shall be the Annual General Meeting.

24.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

25.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members.

26.—The President shall have the power of inviting any person to attend and to take part in the scientific work of the meeting.

27.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a Member, they shall submit the question to a special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

28.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

29.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by R. Percy Smith, M.D., F.R.C.P.

HONORARY MEMBERS.

Professor WUNDT, Leipzig.
,, HITZIG, Halle.
Sir SAMUEL WILKS, Bart., London.
Dr. J. HUGHLINGS JACKSON, London.

CORRESPONDING MEMBERS.

Professor DEJERINE, Paris.
,, ERB, Heidelberg.
,, GOLGI, Pavia.
,, GRASSET, Montpellier.
,, LUCIANI, Rome.
,, MUNK, Berlin.
,, OBERSTEINER, Vienna.
,, A. PICK, Prague.
,, RETZIUS, GUSTAF, Stockholm.
,, RIBOT, Paris.
Dr. WEIR MITCHELL, Philadelphia.
Dr. ALLEN STARR, New York.

LIST OF PRESIDENTS FROM THE FOUNDATION OF THE SOCIETY.

1886 J. HUGHLINGS JACKSON, M.D., F.R.S.
1887 Sir SAMUEL WILKS, Bart., M.D., F.R.S.
1888 Sir JAMES CRICHTON-BROWNE, M.D., F.R.S.
1889 JONATHAN HUTCHINSON, F.R.S.
1890 THOMAS BUZZARD, M.D.
1891 JOHN S. BRISTOWE, M.D., F.R.S.
1892 H. CHARLTON BASTIAN, M.D., F.R.S.
1893 EDWARD A. SCHÄFER, F.R.S.
1894 DAVID FERRIER, M.D., F.R.S.
1895 Sir WILLIAM H. BROADBENT, Bart., M.D., F.R.S.
1896 ALEXANDER HILL, M.A., M.D.
1897 GEORGE H. SAVAGE, M.D.
1898 Sir VICTOR HORSLEY, M.B., F.R.C.S., F.R.S.
1899 W. H. GASKELL, M.D., F.R.S.
1900 AUGUSTUS WALLER, M.D., F.R.S.
1901 W. JULIUS MICKLE, M.D.
1902 HERBERT WILLIAM PAGE, M.A., F.R.C.S.
1903 J. N. LANGLEY, D.Sc., F.R.S.
1904 S. J. SHARKEY, M.D.

LIST OF HUGHLINGS JACKSON LECTURERS.

1897 J. HUGHLINGS JACKSON, M.D., F.R.S.
1900 Geheimrath Professor HITZIG, Halle.
1903 Sir WILLIAM H. BROADBENT, Bart., M.D., F.R.S.

*** Members are requested to communicate with the Secretaries when corrections are necessary.

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EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.	Tr., Treasurer.
Pres., President.	Sec., Secretary.
V.-P., Vice-President.	C., Member of Council.

H.J.L., Hughlings Jackson Lecturer.

ORDINARY MEMBERS.

Elected

1899 ABRAHAMS, BERTRAM, M.B., M.R.C.P., 14, Welbeck Street, W.

1897 ANDERSON, H. KERR, M.D., B.Sc., Whitefield, Great Shelford, Cambs.

1894 ANDRIEZEN, W. LLOYD, M.D., 7, Apsley Terrace, Acton, W.

1903 ARMOUR, D. J., M.B., F.R.C.S., 89, Harley Street, W.

1891 BALLANCE, C. A., M.S., F.R.C.S., 106, Harley Street, W. (C., 1897-1900.)

O.M. BARLOW, Sir THOMAS, Bart., K.C.V.O., M.D., F.R.C.P., 10, Wimpole Street, W. (V.-P., 1904.)

1902 BARNES, A. STANLEY, M.D., 141, Great Charles Street, Birmingham.

1897 BARRATT, J. O. WAKELIN, M.D., F.R.C.S., Bertheaustrasse 6, Göttingen.

O.M. BASTIAN, H. CHARLTON, M.D., F.R.C.P., F.R.S., 8A, Manchester Square, W. (Pres., 1892; V.-P., 1891; C., 1886-89, 1893-95.)

1897 BATTEN, FREDERICK E., M.D., F.R.C.P., 33, Harley Street, W. (Sec., 1903-04.)

1903 BAYLISS, W. M., M.A., D.Sc., F.R.S., St. Cuthbert's, West Heath Road, Hampstead.

O.M. BEACH, FLETCHER, M.B., F.R.C.P., Winchester House, Kingston Hill, Surrey. (C., 1903-04.)

1903 BEDDARD, A. P., M.D., F.R.C.P., 44, Seymour Street, Portman Square.

O.M. BEEVOR, C. E., M.D., F.R.C.P., 135, Harley Street, W. (Tr., 1894-1904; C., 1893.)

1903 BERRY, GEORGE A., M.B., F.R.C.S.Edin., 31, Drumsheugh Gardens, Edinburgh.

1888 BIRT, ERNEST, M.D., West Riding Asylum, Wakefield.

1898 BOLTON, JOSEPH SHAW, M.D., B.S., B.Sc., East Sussex County Asylum, Hellingly, S.O., Sussex.

1896 BOND, C. HUBERT, M.D., D.Sc., London County Colony for Epileptics, Ewell, Surrey.

1899 BOND, J. W., M.D., B.S., 26, Harley Street, W.

1892 BOWLBY, ANTHONY A., C.M.G., F.R.C.S., 24, Manchester Square, W.

Elected

1898 BOYCE, RUBERT W., M.B., F.R.S., University College, Liverpool.

1889 BRADFORD, J. R., M.D., D.Sc., F.R.C.P., F.R.S., 8, Manchester Square, W.

O.M. BRAMWELL, BYROM, M.D., F.R.C.P.Ed., 23, Drumsheugh Gardens, Edinburgh. (C., 1894, 1903-04.)

1899 BRAMWELL, EDWIN, M.B., C.M., M.R.C.P., 23, Drumsheugh Gardens, Edinburgh.

1895 BRAMWELL, JOHN MILNE, M.B., C.M., 15, Stratford Place, W.

1892 BRISTOWE, HUBERT CARPENTER, M.D., Wrington, Somerset.

O.M. BROADBENT, Sir WM. H., Bart., K.C.V.O., M.D., F.R.C.P., F.R.S., 84, Brook Street, W. (Pres., 1895; V.-P., 1893; C., 1886-87, 1896.)

1894 BRODIE, T. GRIGOR, M.D., 55, Marlborough Mansions, West Hampstead, N.W.

1897 BROWN, H. H., M.D., F.R.C.S., 22, Museum Street, Ipswich.

1888 BROWN, SANGER, M.D., 100, State Street, Chicago.

O.M. BRUCE, ALEX., M.D., F.R.C.P.Ed., 8, Ainslie Place, Edinburgh. (C., 1895.)

O.M. BRUCE, J. MITCHELL, M.D., F.R.C.P., 23, Harley Street, W.

1898 BRUCE, SAMUEL NOBLE, M.R.C.S., 15, Queensboro' Terrace, Hyde Park, W.

O.M. BRUNTON, Sir T. LAUDER, M.D., F.R.C.P., F.R.S., 10, Stratford Place, W. (C., 1896-98.)

1895 BRYAN, FREDERICK, M.B., Horton House, Horton, Epsom.

1895 BRYANT, JOHN HENRY, M.D., F.R.C.P., 8, Mansfield Street, Portland Place, W.

1903 BUBB, WILLIAM, M.R.C.S., L.R.C.P., County Asylum, Powick, Worcester.

1890 BURY, JUDSON SYKES, M.D., F.R.C.P., 10, St. John Street, Manchester.

1900 BUZZARD, E. FARQUHAR, M.D., M.R.C.P., 33, Harley Street, W.

O.M. BUZZARD, THOMAS, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.-P., 1888-89; C., 1886-87, 1891-95.)

1898 CALEY, H. A., M.D., F.R.C.P., 24, Upper Berkeley Street, W.

1903 CAMPBELL, A. W., M.D., County Asylum, Rainhill, Lancs.

1898 CAMPBELL, HARRY, M.D., F.R.C.P., 23, Wimpole Street, W.

1887 CATTELL, J. MCK., M.A., Ph.D., Columbia College, New York, U.S.A.

1899 CHAMBERS, JAMES, M.A., M.D., The Priory, Roehampton.

1891 CLARKE, J. MICHELL, M.D., F.R.C.P., 28, Pembroke Road, Clifton, Bristol. (C., 1903-04.)

1903 CLOUSTON, T. S., M.D., F.R.C.P.Ed., Royal Asylum, Morningside, Edinburgh.

1892 COLE, ROBERT HENRY, M.D., M.R.C.P., 48, Upper Berkeley Street, Portman Square.

1900 COLE, SYDNEY JOHN, M.O. Wilts County Asylum, Devizes.

1899 COLLIER, J. S., M.D., B.Sc., F.R.C.P., 57A, Wimpole Street, W.

1890 COLMAN, W. S., M.D., F.R.C.P., 9, Wimpole Street, W. (Sec., 1899-1902. C., 1903-04.)

1890 CORNER, HARRY, M.D., Brook House, Southgate, Middlesex.

1899 COUPLAND, SIDNEY, M.D., F.R.C.P., 16, Queen Anne Street, W.

Elected

1901 Cox, Joshua John, M.D., St. Ronan's, Clarendon Road, Eccles, Manchester.

1901 Craig, Maurice, M.D., M.R.C.P., Bethlem Royal Hospital, S.E.

1898 Darley-Hartley, W., M.D., M.R.C.P., Main Road, Sea Point, Cape Town, South Africa.

1895 Davidson, Andrew, M.D., Callan Park, Sydney, N.S.W.

1897 Dawson, William R., M.D., F.R.C.P.I., Farnham House, Finglas, co. Dublin, Ireland.

1898 Dodd, Henry Work, F.R.C.S., 136, Harley Street, W.

O.M. Dreschfeld, Julius, M.D., F.R.C.P., 3, St. Peter's Square, Manchester. (V.-P., 1898-99.)

O.M. Drummond, David, M.D., 6, Saville Place, Newcastle-on-Tyne.

1903 Dudgeon, L. S., M.R.C.P., 6, Powis Gardens, London, W.

1892 Dupuy, Eugene, M.D., 53, Avenue Montaigne, Paris.

1901 Edridge-Green, F. W., M.D., F.R.C.S., 14, Welbeck Street, W.

1899 Fawcett, John, M.D., F.R.C.P., 26, St. Thomas Street, S.E.

O.M. Ferrier, David, M.D., F.R.C.P., F.R.S., 34, Cavendish Square, W. (Pres., 1886; V.-P., 1892-3; C., 1886, 1890-95.)

1903 Flashman, J. F., M.B., Ch.M., B.Sc., Totnes, Temple Street, Petersham, Sydney, Australia.

1903 Fleming, Robert, M.D., F.R.C.P.Ed., 10, Chester Street, Edinburgh.

1903 Fletcher, H. Morley, M.D., F.R.C.P., 98, Harley Street, W.

O.M. Gaskell, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs. (Pres., 1899; V.-P., 1897-98; C., 1896, 1900.)

1903 Gibson, G. A., M.D., D.Sc., F.R.C.P.Ed., 3, Drumsheugh Gardens, Edinburgh.

O.M. Godlee, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1902 Goldschmidt, Oscar Bernard, M.B., Bishopstone House, Bedford.

1899 Goodall, Edwin, M.D., F.R.C.P., Joint Counties Asylum, Carmarthen.

1898 Goodliffe, Jno. Henry, M.D.Aberd., 802, High Road, Lower Tottenham.

1897 Gossage, Alfred Milne, M.B., F.R.C.P., 54, Upper Berkeley Street, W.

1892 Gotch, Francis, M.A., F.R.S., The Lawn, Banbury Road, Oxford.

1893 Grant, J. Dundas, M.D., F.R.C.S., 18, Cavendish Square, W.

O.M. Gunn, R. Marcus, M.B., F.R.C.S., 54, Queen Anne Street, W. (C., 1899-1900.)

1895 Guthrie, Leonard, M.D., F.R.C.P., 15, Upper Berkeley Street, W. (C., 1904.)

1902 Hall, W. Hamilton, M.R.C.S., Fordcombe, Tunbridge Wells.

1893 Halliburton, William Dobinson, M.D., F.R.C.P., F.R.S., Church Cottage, 17, Marylebone Road, N.W.

O.M. Hamilton, D. J., M.B., F.R.S.E., University, Aberdeen.

1898 Hanbury, William R., M.R.C.S., L.R.C.P., West Ham Borough Asylum, Goodmayes, Ilford.

1897 Harris, Wilfred J., M.D., M.R.C.P., 37, Queen Anne Street, W.

Elected

1898 HASLETT, JOHN HANFIELD, M.R.C.S., L.R.C.P., Halliford House, Sunbury-on-Thames.

1891 HAWKINS, HERBERT PENNELL, M.D., F.R.C.P., 56, Portland Place, W.

O.M. HAYCRAFT, J. B., M.D., D.Sc., F.R.S.E., University College of South Wales, Cardiff.

1891 HEAD, HENRY, M.D., F.R.C.P., F.R.S., 143, Harley Street, W. (C., 1900; Sec., 1901-1904.)

O.M. HILL, ALEX., M.A., M.D., Downing Lodge, Cambridge. (Pres., 1896; V.-P., 1895; C., 1892-94.)

1903 HOLMES, GORDON M., M.B., B.Ch., National Hospital, Queen Square, W.C.

O.M. HORSLEY, Sir VICTOR, F.R.C.S., F.R.S., 25, Cavendish Square, W. (Pres., 1898; V.-P., 1896-97; C., 1892-93, 1899.)

1903 HOWLAND, GOLDWIN, W., M.B., M.R.C.P., National Hospital.

1889 HUGGARD, WILLIAM R., M.D., F.R.C.P., Davoz Platz, Switzerland.

1896 HULME, GEORGE FREDERICK, M.B., C.M., South View, Montague Road, Felixstowe.

1889 HUMPHRY, LAURENCE, M.D., M.R.C.P., Lensfield, Cambridge.

1903 HUNTER, WALTER K., M.D., D.Sc., 7, Woodside Place, Glasgow.

1901 HUNTER, WILLIAM, M.B., C.M., Government Bacteriological Laboratory, Hong Kong.

1903 HUNTER, WILLIAM, M.D., F.R.C.P., 103, Harley Street, W.

O.M. HUTCHINSON, JONATHAN, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889; V.-P., 1887-88; C., 1886, 1890.)

1889 HYSLOP, T. B., M.D., C.M., Bethlem Royal Hospital, Lambeth, S.E.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.C.P., F.R.S., 3, Manchester Square, W. (Pres., 1886; H.J.L., 1897; C., 1887, 1892.). Hon. Memb., 1904.

O.M. JESSOP, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1895 JOHNSTON, GEORGE, M.D., M.R.C.P., 3, Montagu Place, Montagu Square, W.

1903 JONES, A. E., M.B., B.Sc., University College Hospital, Gower Street.

1892 JONES, HENRY LEWIS, M.D., F.R.C.P., 143, Harley Street, W.

1894 JONES, ROBERT, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.

1902 KELYNACK, T. N., M.D., M.R.C.P., 120, Harley Street, W.

1894 KIDD, PERCY, M.D., F.R.C.P., 60, Brook Street, W. (C., 1904.)

1896 LANGDON, F. W., M.D., Cincinnati, Ohio, U.S.A.

1898 LANGDON-DOWN, REGINALD L., M.B., M.R.C.P., 47, Welbeck Street, W.

O.M. LANGLEY, J. N., M.A., D.Sc., F.R.S., Hedgerley Lodge, Madingley Road, Cambridge. (Pres., 1903; V.-P., 1901-1902; C., 1898-99, 1904.)

1888 LAWFORD, J. B., M.D., F.R.C.S., 99, Harley Street, W. (C., 1902-3.)

O.M. LEES, D. B., M.D., F.R.C.P., 22, Weymouth Street, W. (C., 1901-1903.)

1897 LEWIS, H. WOLSELEY, M.R.C.S., L.R.C.P., London County Asylum, Banstead, Surrey.

1904 LINDSAY, J. A., M.D., F.R.C.P., 13, College Square E., Belfast.

1901 LITTLE, E. GRAHAM, M.D., M.R.C.P., 61, Wimpole Street, W.

Elected

1897 LOEWENTHAL, MAX, M.D., M.R.C.P., 126, Princes' Road, Liverpool.
1899 LORD, JOHN R., M.B., C.M., London County Asylum, Bexley, Kent.

1898 MCALDOWIE, ALEXANDER M., M.D., 6, Brook Street, Stoke-on-Trent.
1898 MACCORMAC, JOHN M'GEE, M.D., 31, Victoria Place, Belfast.
1891 MACDONALD, PETER WILLIAM, M.D., C.M., The County Asylum, Dorchester, Dorset.
1902 MCDOUGALL, WM., M.B., Weald Mount, Haslemere.
1902 MACKENZIE, JAMES, M.D., Bank Parade, Burnley.
O.M. MACKENZIE, Sir STEPHEN, M.D., F.R.C.P., 31, Cavendish Square, W. (C., 1896-98.)
1896 MACKINTOSH, ASHLEY W., M.A., M.D., 9, Bon Accord Square, Aberdeen.
1903 MACNAMARA, E. D., M.B., B.C., 45, Campden House Road, W.
O.M. MAGUIRE, ROBERT, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893-96.)
O.M. MANN, J. DIXON, M.D., F.R.C.P., 16, St. John Street, Manchester.
1902 MANTLE, ALFRED, M.D., M.R.C.P., Savile Place, Halifax.
1901 MARRIS, W. A., M.D., Springfield Road, King's Heath, Birmingham.
1894 MARTIN, SIDNEY, M.D., F.R.C.P., F.R.S., 10, Mansfield Street, W.
1891 MAY, W. PAGE, M.D., F.R.C.P., Helouan, Cairo, Egypt.
1890 MENZIES, WILLIAM FRANCIS, M.D., B.Sc., M.R.C.P., Cheddleton Asylum, Leek, Staffordshire.
O.M. MERCIER, C., M.B., M.R.C.P., Flower House, Southend, Catford, S.E. (C., 1899-1902.)
1897 MEYER, ADOLF, M.D., LL.D., Pathological Institute, New York State Hospital, Ward's Island, N.Y., U.S.A.
O.M. MICKLE, W. JULIUS, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (Pres., 1901; V.-P., 1900; C., 1895-97, 1902-4.)
1895 MOORE, NORMAN, M.D., F.R.C.P., 94, Gloucester Place, W.
O.M. MORRIS, MALCOLM, F.R.C.S.Ed., 8, Harley Street, W.
1902 MORRISON, C. S., L.R.C.P.E., County and City Asylum, Hereford.
1888 MOTT, F. W., M.D., F.R.C.P., F.R.S., 25, Nottingham Place, W. (Sec., 1894-98; C., 1899-1902.)
1901 MOULD, GEORGE WM., M.R.C.S., Cheadle Royal Asylum, Manchester.
1899 MOULD, GILBERT E., M.R.C.S., L.R.C.P., The Grange, nr. Rotherham.
1894 MURRAY, GEORGE R., M.A., M.D., F.R.C.P., 11, Ellison Place, Newcastle-on-Tyne.
1889 MURRAY, H. MONTAGUE, M.D., F.R.C.P., 25, Manchester Square, W.

1904 NEILD, NEWMAN, M.D., Richmond Hill, Clifton, Bristol.
O.M. NETTLESHIP, EDWARD, F.R.C.S., Shottermill, Haslemere, Surrey.
1902 NEWINGTON, HAYES, F.R.C.P.Ed., The Gables, Ticehurst, Sussex.
O.M. NICOLSON, DAVID, C.B., M.D., M.R.C.P.Ed. Balgownie, Guildford, Surrey.
1888 NIERMEYER, J. H. H., M.D., Amsterdam.
1904 NOLAN, M. J., L.R.C.P., L.R.C.S.I., Down District Asylum, Downpatrick.
1888 NORMAN, CONOLLY, F.R.C.P.I., F.R.C.S.I., St. Dymphna's, N. Circular Road, Dublin.

Elected

1887 OGILVIE, GEORGE, M.B., M.R.C.P., 22, Welbeck Street, W.

1887 OLIVER, THOMAS, M.D., F.R.C.P., 7, Ellison Place, Newcastle-on-Tyne.

O.M. ORANGE, WM., C.B., M.D., F.R.C.P., Oakhurst, Godalming, Surrey.

O.M. ORMEROD, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895-97.)

1899 ORR, DAVID, M.D., County Asylum, Prestwich, Manchester.

O.M. PAGE, HERBERT WILLIAM, M.A., F.R.C.S., 146, Harley Street, W. (Pres., 1902; V.-P., 1900-1; C., 1891-92, 1903-04.)

1903 PALMER, FREDK. S., M.D., M.R.C.P., 7, Gordon Place, Gordon Square, London.

1895 PARKINSON, JOHN PORTER, M.D., M.R.C.P., 57, Wimpole Street, W.

1901 PARSONS, JOHN HERBERT, M.B., B.Sc., 28, Great Ormond Street, W.C.

1901 PASSMORE, E. S., M.D., M.R.C.P., Croydon Asylum, Warlingham, Surrey.

1896 PATERSON, DONALD ROSE, M.D., M.R.C.P., 15, St. Andrew's Crescent, Cardiff.

1899 PIPER, FRANCIS P., M.B., M.R.C.S., L.R.C.P., King's Leigh, Tankerton, Whitstable.

1888 PITT, G. N., M.D., F.R.C.P., 15, Portland Place, W. (C., 1903-04).

O.M. PURVES, W. LAIDLAW, M.D., 20, Stratford Place, W.

1899 RAMBAUT, DANIEL F., M.D., B.Ch., County Asylum, Bicton Heath, Shrewsbury.

1894 RANSOM, WILLIAM B., M.D., F.R.C.P., The Pavement, Nottingham.

1890 RAYNER, HENRY, M.D., M.R.C.P.Ed., 16, Queen Anne Street, W.

O.M. REID, E. W., M.B., F.R.S., University College, Dundee.

1897 REISSMANN, CHARLES H., M.B., B.Sc., St. Peter's, Adelaide, South Australia.

1893 RENNIE, GEORGE E., M.D., M.R.C.P., College Street, Sydney.

1895 REYNOLDS, ERNEST S., M.D., F.R.C.P., 2, St. Peter's Square, Manchester.

1891 RIVERS, W. H. R., M.A., M.D., F.R.C.P., St. John's College, Cambridge. (C., 1897-98.)

1898 ROBERTSON, WILLIAM FORD, M.D., 7, Hill Square, Edinburgh.

1892 ROWE, EDMUND LEWIS, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.

1901 ROWS, R. G., M.D., County Lunatic Asylum, Lancaster.

1892 RUFFER, MARC ARMAND, M.D., Ramleh, Egypt.

1903 RUSSELL, A. E., M.D., M.R.C.P., 9, Wimpole Street, W.

1889 RUSSELL, J. S. RISIEN, M.D., F.R.C.P., 44, Wimpole Street, W. (Sec., 1896-1900; C., 1901-1904.)

1899 RUSSELL, J. W., M.D., M.R.C.P., 72, Newhall Street, Birmingham.

1901 SALAMAN, REDCLIFFE N., M.A., M.B., Frognal End, Frognal Gardens, Hampstead.

O.M. SANDERSON, Sir J. BURDON, Bart., M.D., F.R.C.P., F.R.S., Banbury Road, Oxford.

O.M. SAVAGE, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square. (Pres., 1897; V.-P., 1895; C., 1886-90, 1898.)

Elected

1888 SAVILL, T. D., M.D., M.R.C.P., 60, Upper Berkeley Street, W.

O.M. SCHÄFER, E. A., F.R.S., University of Edinburgh. (Pres., 1893; V.-P., 1892; C., 1886-98.)

1901 SCHORSTEIN, GUSTAVE, M.D., F.R.C.P., 11, Portland Place, W.

O.M. SEMON, Sir FELIX, C.V.O., M.D., F.R.C.P., 39, Wimpole Street, W. (C., 1899-1901.)

1898 SEWARD, WM. JOSEPH, M.B., Colney Hatch Asylum, N.

O.M. SHARKEY, S. J., M.D., F.R.C.P., 22, Harley Street, W. (Pres., 1904; V.-P., 1902-3.; C., 1899-92.)

O.M. SHERRINGTON, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (C., 1892-95.)

1894 SHUTTLEWORTH, GEORGE E., M.D., Ancaster House, Richmond.

1898 SIMPSON, FRANCIS O., L.R.C.P., M.R.C.S., Rainhill Asylum, Liverpool.

1900 SINGER, H. D., M.D., M.R.C.P., St. Thomas's Hospital, S.E.

1895 SMITH, F. J., M.D., F.R.C.P., 138, Harley Street, W.

1887 SMITH, R. PERCY, M.D., F.R.C.P., 36, Queen Anne Street, W. (Editor of "Brain," 1901-1904.)

1895 SPICER, W. T. HOLMES, M.B., F.R.C.S., 5, Wimpole Street, W.

1901 STAFFORD, WILLIAM, M.B., 110, Mansfield Road, Nottingham.

1895 STANLEY, DOUGLAS, M.D., M.R.C.P., 21, Broad Street, Birmingham.

1895 STANSFIELD, THOS. E. K., M.B., London County Asylum, Bexley, Kent.

1892 STARLING, ERNEST HENRY, M.D., F.R.C.P., F.R.S., 8, Park Square West, Regent's Park, N.W. (C., 1904.)

1901 STEELL, GRAHAM, M.D., F.R.C.P., 96, Mosley Street, Manchester.

1897 STEWART, PURVES, M.A., M.D., M.R.C.P., 7, Harley Street, W.

1903 STEWART, T. GRAINGER, M.B., B.Ch., M.R.C.P., National Hospital, Queen Square, W.C.

1897 STIRLING, WM., M.D., D.Sc., Owens College, Manchester.

1897 STODDART, WM. H., M.D., M.R.C.P., Bethlem Royal Hospital, Lambeth, S.E.

1902 STONE, W. G., M.D., 93, Denmark Hill, S.E.

1887 SUCKLING, C. W., M.D., M.R.C.P., 103, Newhall Street, Birmingham.

1902 SULLIVAN, WM. CHARLES, M.D., H.M. Prison, Pentonville, N.

1904 SUTHERLAND, GEO. ALEXANDER, M.D., F.R.C.P., 73, Wimpole Street, W.

1888 SYERS, HENRY W., M.D., M.R.C.P., 75, Wimpole Street, W.

1898 TAYLOR, F. R. P., M.D., East Sussex Asylum, Hellingly, Sussex.

1891 TAYLOR, JAMES, M.D., F.R.C.P., 49, Welbeck Street, W. (C., 1900-3.)

1898 THOMSON, H. CAMPBELL, M.D., F.R.C.P., 34, Queen Anne Street, W.

1889 THORBURN, WM., F.R.C.S., Oxford Lodge, Victoria Park, Manchester. (C., 1901-1903.)

1892 TITCHENER, E. B., B.A., Ph.D., Cornell Heights, Ithaca, N.Y.

O.M. TOOTH, H. H., C.M.G., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95; C., 1896-99.)

1892 TREVELYAN, E. F., M.D., B.Sc., F.R.C.P., 40, Park Square, Leeds.

1892 TUCKEY, CHARLES LLOYD, M.D., 88, Park Street, Grosvenor Square, W.

1892 TUKE, Sir J. BATTY, M.D., F.R.C.P.Ed., F.R.S.Ed., 20, Charlotte Sq., Edinburgh. (V.P. 1903-04.)

Elected

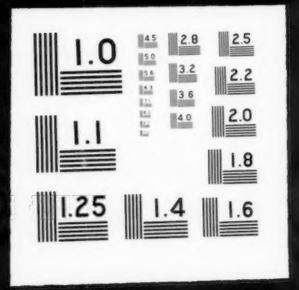
1891 TUKE, THOMAS SEYMOUR, M.B., Chiswick House, Chiswick.
1891 TURNER, WILLIAM ALDREN, M.D., F.R.C.P., 18, Harley Street.
1898 TURNEY, HORACE G., M.D., F.R.C.P., 68, Portland Place, W.
O.M. TWEEDY, JOHN, F.R.C.S., 100, Harley Street, W.

1901 VINCENT, SWALE, M.B., Physiological Laboratory, University College, Cardiff.
1888 VOORTHUS, J. A., M.D., Apeldoorn, 542, Loulaan, Netherlands.

O.M. WALLER, AUGUSTUS, M.D., F.R.S., 32, Grove End Road, N.W. (Pres. 1900; V.-P., 1899; C., 1894-97, 1901.)
O.M. WARNER, FRANCIS, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.
1902 WARRINGTON, W. B., M.D., M.R.C.P., 69, Rodney Street, Liverpool.
1904 WATSON, CHALMERS, M.B., F.R.C.P.E., 22, Coates Crescent, Edinburgh.
O.M. WATTEVILLE, A. DE, M.A., M.D., B.Sc., 30, Welbeck Street, W. (C., 1890-95, 1896-1902; Sec., 1886-89; Editor of "Brain," 1880-1900.)
1897 WEBER, F. PARKES, M.D., F.R.C.P., 19, Harley Street, W.
1897 WEST, SAMUEL, M.D., F.R.C.P., 15, Wimpole Street, W.
1897 WHITE, ERNEST W., M.B., M.R.C.P., City of London Asylum, Dartford.
O.M. WHITE, W. HALE, M.D., F.R.C.P., 65, Harley St., W. (C., 1897-1902.)
1894 WHITING, ARTHUR J., M.D., M.R.C.P., 142, Harley Street, W.
1889 WIGLESWORTH, JOSEPH, M.D., F.R.C.P., County Asylum, Rainhill, Lancashire.
1900 WILLIAMS, LEONARD, M.D., M.R.C.P., 8, York Street, Portman Square, W.
1894 WILLIAMSON, RICHARD T., M.D., F.R.C.P., 2, St. Peter's Square, Manchester.
1896 WILSON, ALBERT, M.D., 1, Belsize Park, N.W.
1903 WILSON, G. R., M.D., M.R.C.P.Ed., 8, Rutland Square, Edinburgh.
1896 WOOD, GUY M., M.B., 6, Woburn Square, W.C.
1889 WOOD, T. OUTTERSON, M.D., F.R.C.P.Ed., 40, Margaret Street, Cavendish Square, W.
1892 WOODHEAD, G. SIMS, M.D., F.R.C.P.Ed., 6, Scroope Terrace, Cambridge.
1899 WRIGHT, HAMILTON, M.D., Pathological Institute, Selangor, Straits Settlement.

1898 YOUNG, ROBERT ARTHUR, M.D., B.Sc., M.R.C.P., 41, Wimpole St., W.

RESOLUTION CHART



100 MILLIMETERS

INSTRUCTIONS Resolution is expressed in terms of the lines per millimeter recorded by a particular film under specified conditions. Numerals in chart indicate the number of lines per millimeter in adjacent "T-shaped" groupings.

In microfilming, it is necessary to determine the reduction ratio and multiply the number of lines in the chart by this value to find the number of lines recorded by the film. As an aid in determining the reduction ratio, the line above is 100 millimeters in length. Measuring this line in the film image and dividing the length into 100 gives the reduction ratio. Example: the line is 20 mm. long in the film image, and $100/20 = 5$.

Examine "T-shaped" line groupings in the film with microscope, and note the number adjacent to finest lines recorded sharply and distinctly. Multiply this number by the reduction factor to obtain resolving power in lines per millimeter. Example: 7.9 group of lines is clearly recorded while lines in the 10.0 group are not distinctly separated. Reduction ratio is 5, and $7.9 \times 5 = 39.5$ lines per millimeter recorded satisfactorily. $10.0 \times 5 = 50$ lines per millimeter which are not recorded satisfactorily. Under the particular conditions, maximum resolution is between 39.5 and 50 lines per millimeter.

Resolution, as measured on the film, is a test of the entire photographic system, including lens, exposure, processing, and other factors. These rarely utilize maximum resolution of the film. Vibrations during exposure, lack of critical focus, and exposures yielding very dense negatives are to be avoided.